" FUNCTION AND STRUCTURE IN THE VISUAL PATHWAY".

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CONTENTS.

"FUNCTIONS AND STRUCTURE IN THE VISUAL PATHWAYS"

ABSTRACT

1. Small lesions have been made in the cat lateral geniculate nucleus. The resulting degeneration has been traced in the neocortex using the Nauta stain. A projection, which is strictly ipsilateral to the lesion, is described to the visual areas I and II (as defined by TALBOT, 1942 and BILGE, SENEVIRATNE & WHITTERIDGE, 1963). A third projection was found to the lateral edge of the middle suprasylvian gyrus. More medial lesions which included the medial interlaminar nucleus of the lateral geniculate nucleus produced degeneration in Visual area III (HUBEL & WIESEL, 1965; WHITTERIDGE, 1966) in the bottom of the splenial sulcus and thirdly, on the middle suprasylvian gyrus.
2. Similar methods in the monkey have shown only one projection from the LGN; this is to the striate area.
3. Small lesions have been made in various cortical visual areas of the cat. A study of the Nauta degeneration has shown that these cortical areas are reciprocally connected. The densest projection is from the medial to the more lateral areas. Reciprocal connections exist with the other hemisphere, these are sparse except for the regions which represent the vertical meridian of the visual field. Some evidence has been found for a projection from the middle suprasylvian gyrus to the anterior sigmoid gyrus.
4. Photically evoked electrical responses mediated by the corpus callosum have been studied in the cat lateral gyrus after the optic tract had been cut on that side. Responses could only be obtained from a narrow strip corresponding to the vertical meridian of the visual field (Visual I/Visual II boundary). The strip spread laterally in the region of the area centralis and was traced for

a short distance in the region corresponding to the vertical meridian of Visual III. Responses could only be elicited if the photic stimulus was close to the vertical meridian. Single units were observed, these were simple units (HUBEL & WIESEL, 1960). The responses on the VisualI/Visual II boundary could be abolished temporarily by cooling the corresponding point of the other hemisphere or the corpus callosum.

5. The discreetness of the connections observed with electrophysiological methods is contrasted with the diffuseness seen with the Nauta method. Similarities between the cat and monkey visual areas and between the cat supraylvian and the monkey temporal regions are discussed.

6. An attempt was made to differentiate behaviourally between the A or A1 and the B laminae of the cat $_{\Lambda}^{LGN}$ by cutting one optic tract and testing monocularly. No difference was found in the absolute threshold to light or the ability to discriminate between patterns which lacked edges. These were the two most likely deficits suggested by previous electrphysiological studies. (SENEVIRATNE & WHITTERIDGE, 1962).

7. Some neurones were found to persist in all laminae of the cat lateral geniculate nucleus inspite of removal of all the neocortical visual projections.

REVIEW OF THE LITERATURE

The experimental results will deal with the projections from the thalamus to the cortical visual areas, and the interconnection of these visual areas in the cat. The literature has been reviewed with this in mind and will deal largely with the visual pathways in the cat.

TRACING NERVE PATHWAYS

As the reliability of the experiments to be discussed depends on their method, it seems worthwhile describing and criticising these techniques in general terms at the outset. It is appreciated that some of the points are elementary but this does not belittle their importance. As will be seen, they have been ignored by a number of workers. These techniques will be described under the following headings :-

1. Anatomical methods.
   a) Anterograde - Marchi, Nauta, Glees
   b) Transynaptic atrophy
   c) Retrograde - Nissl, Nauta
   d) Golgi
   e) Electron microscopy

2. Physiological methods.

Stimulus	Recording electrode
- natural	- gross
- shock	- micro
- strychnine	- intracellular

1. Anatomical Methods

When the axon of a neurone is destroyed the distal portion of the cut axon breaks down (anterograde reaction). The cell body, and in particular its Nissl substance, undergoes a series of changes (retrograde reaction). Appropriate stains will reveal these changes, which are usually first detectable in the peripheral portion of the axon. So, by cutting a nerve, its cell body and its peripheral axon can be found.

Obviously in making the lesion other structures must not be damaged. I f the lesion is made by an inserted electrode, damage due to the track must be allowed for. The lesion must not damage adjacent nuclear groups and the destination of any fibres of passage which run through the nucleus should be known. With a cortical lesion care should be taken that it does not extend into the white matter cutting into fibres belonging to other areas and that there should be no incidental damage due to the operative procedures.

a) Anterograde Methods

(i) The Marchi method is an osmium tetroxide stain which shows a characteristic picture of degenerating myelin globules. This has now largely been superseded by the Nauta stain because Marchi does not stain non-myelinated nerve fibres and this has the particular disadvantage that crucial knowledge of preterminal distribution can not be ascertained, particularly in the lightly myelinated regions. It is seldom obvious that neighbouring droplets are derived from the same fibre and this may make distinction from artefact difficult.

(ii) Nauta-Gygax. In this method normal fibres are suppressed leaving degenerating fibres densely stained with silver as a series of intensely black dashes and dots against a pale brown or yellow

background. Terminal boutons can seldom be seen but the whole axon distal to the lesion and the preterminal fibres are stained as the silver is attached to the membranes of axoplasmic organelles as well as of myelin (LUND & WESTRUM, 1966). If the dots are arranged in a linear series it is usually considered that they are fibres of passage in contrast to preterminal degeneration where short arcs are formed around a cell, (see BERESFORD, 1961, who prefers the term undirected degeneration in this last case). It may not always be possible to make these distinctions with certainty.

(iii) Some degenerating boutons undergo neurofibrillar hypertrophy. Silver is deposited on these neurofibrils with the Glees method (LUND & WESTRUM, 1966). In some parts of the nervous system (e.g. cortex) neurofilaments do not appear during degeneration and the Glees method is of no use (see GUILLERY, 1965). The swollen degenerated boutons must be looked for amongst a mesh of other stained neuronal components including fragments of degenerating axons which may mimic degenerating boutons. Comparison should always be made with normal boutons from the same site (HAYHOW, 1959).

The relative merits of the Marchi, Nauta and Glees methods have been discussed by GLEES & NAUTA (1955), EVANS & HAMLYN (1956) and BOWSHER, BRODAL & WALBERG (1960).

b) Transynaptic atrophy occurs in only certain special situations such as the LGN. When the afferent nerve is cut the postsynaptic neurones atrophy and may even disappear. There is considerable species variation in the degree and rate at which the atrophy occurs and it is more rapid and severe in immature animals. COOK, WALKER & BARR (1951), MATTHEWS, COWAN & POWELL (1960), MATTHEWS (1964), KUPFER & PALMER (1964).

c) Retrograde degeneration

After an initial swelling of the cell there is loss of Nissl substance, the cell shrinks and may finally disappear. There is an increase in the number of glial cells. In contrast to the Nauta method, these changes are less striking and will not be apparent unless many neurones are affected.

In some situations retrograde degeneration does not occur. DAITZ & POWELL (1954) did not find retrograde degeneration in hippocampal neurones after they had cut the fornix. It is suggested that if the axon divides into a thick and a fine branch that retrograde degeneration will not be seen if the fine branch alone is cut. The thick one is capable of 'sustaining' the cell. Degeneration will follow damage to the thick branch but may be more marked if both are cut (ROSE & WOOLSLEY, 1958). Retrograde degeneration has also been described in Nauta stained material. This occurs at a later stage than the anterograde degeneration and is characterised by a diffuse, black, granular appearance which does not show a pericellular relationship (GUILLERY, 1959; CRAGG, 1962; POWELL & COWAN, 1964). In an 11 - 18 day old kitten GRANT (1965) has described Nauta staining of apparently degenerating dendrites which could be confused with typical anterograde degeneration.

d) The validity of the Golgi method depends on the complete staining of a small but representative propertion of cells (SHOLL, 1953). It is difficult to trace the ramifications for any distance; neurones may be incompletely stained; immature animals are often used and it is conceivable that their nerve pathways are not yet fully established.

e) Electron microscopy may have advantages for demonstrating degenerating components of a neurone but because of the small area sampled

degeneration will only be found if it is widespread. For the same reason, it is not feasible to trace neurones over any distance.

2. Physiological Methods

Stimulating techniques

A neurone may be stimulated electrically at one end and recorded from at the other, the impulse being propogated either orthodromically or antidromically, or the peripheral end may be stimulated naturally through its receptor. A bundle of nerve fibres may contain some fibres which are excitatory and others which are inhibitory on the recording site. Stimulation of the whole nerve bundle may then produce a false negative result. Thus HUBEL & WIESEL (1959) were able to activate far more cells in the visual cortex using an appropriately orientated dark bar or slit of light than with the whole visual field illuminated.

There is at least the theoretical possibility that a synchronous volley of impulses might be sufficient to fire a group of neurones which would not be fired by the natural asynchronous barrage of impulses.

The effect of small stimulus voltages will depend on the shape of the electrodes and the proximity and thresholds of the various neurones in the vicinity (BUREŠ, 1960). The onus is upon the experimenter to show that the stimulus has not spread. Although electrical stimulation may be useful for latency measurements as receptor variability is removed, it does have the serious disadvantages which have been discussed when used for tracing pathways.

It was originally held that when strychnine is applied locally to the grey matter of the central nervous system strychnine spikes could be recorded not only at the strychninised focus, but also at

other distant points which were directly connected by nerve fibres to the strychninised focus. However, the method has serious limitations. Not all neurones are activated by strychnine so that false negative results may occur (DOW, 1938; FRANKENHAEUSER, 1951; WALL & HORWITZ, 1951). The latter workers have also shown that synchronised barrage of impulses could cross some synapses and that neurones situated at some distance from the strychninised area might be stimulated because of the enhanced electrotonic effects.

Recording techniques

A large recording electrode will be influenced by the electrical activity over a wide area. This effect may be minimised by bipolar electrodes (e.g. INGVAR & HUNTER, 1955, who compared monopolar and bipolar electrodes) and a push-pull amplification. Microelectrodes with tips around 1μ in diameter will record activity from single units (cell bodies or sometimes axons). Even finer electrodes can be inserted into the larger cells. The last two types of electrodes have disadvantages. They give information about only a small select sample of neurones. The fine extracellular electrodes are influenced by background 'noise' which may obscure the potential being studied.

A small response may not be apparent above the random level of neuronal activity. This background may be electronically averaged over a number of stimulus presentations allowing the consistent response to be seen (DAWSON, 1954).

Again, the experimenter should show that his electrodes are recording only from the desired area. If stimulation of point A results in a response at point B care should be taken to establish if any interneurones are present; the pathway may not be the most direct. A synapse between the two points may be difficult to establish as the extra delay may be small compared with the long latency

between A and B. It may be difficult to decide where to measure the latency on the record if the rise time of the wave is slow. On the other hand delay may be introduced without a synapse if the fibre branches and becomes thinner. It is not justifiable to measure latencies between the point of stimulation and recording as under these circumstances the latency measured varies with stimulus strength (MILEDI, 1957). However, the error introduced is only significant when latencies of a few milliseconds are being measured.

The pathway may be blocked reversibly by the injection of some agent, by local cooling or irreversibly, by making a lesion. This may be a useful method for demonstrating that a nucleus or other part of the brain is an integral part of the chain linking two points.

Whether the animal is well, shocked, unanaesthetized, cerveau isolé or encephale isolé, or anaesthetized with a barbiturate, chloralose or other drug is important. ROBERTSON (1965) has shown that the behaviour of cells in the cerveau isolé cat visual cortex changes when nembutal is injected. The responses from LGN lamina B cells are abolished by barbiturates (SENEVIRATNE, 1962). These are just a couple of examples.

B. THE RETINA

1. ANATOMY

Light energy is received by the rods and cones. After crossing the intra-retinal synapses the nerve impulses are finally transmitted to the brain by the optic nerve. The ganglion cells of the retina are the cell bodies of the fibres which compose the optic nerve. Their distribution has been quantitatively described by STONE (1965) in the cat using whole mounts of the retina stained with methylene blue. There is a great increase in the cell density (chiefly of small diameter cells 8 - 10μ) at the area centralis. Blood vessels and nerve fibres converge upon but do not pass across this area. A band of ganglion cells of relatively high density spreads out from this area approximately horizontally into nasal and temporal retinae. STONE (1966) has described an area of overlap between nasal and temporal portions of the retinae as defined by their retrograde degeneration appearance after unilateral optic tract section. Furthermore, 25% of temporal cells project contralaterally. This can best be understood from this diagram.

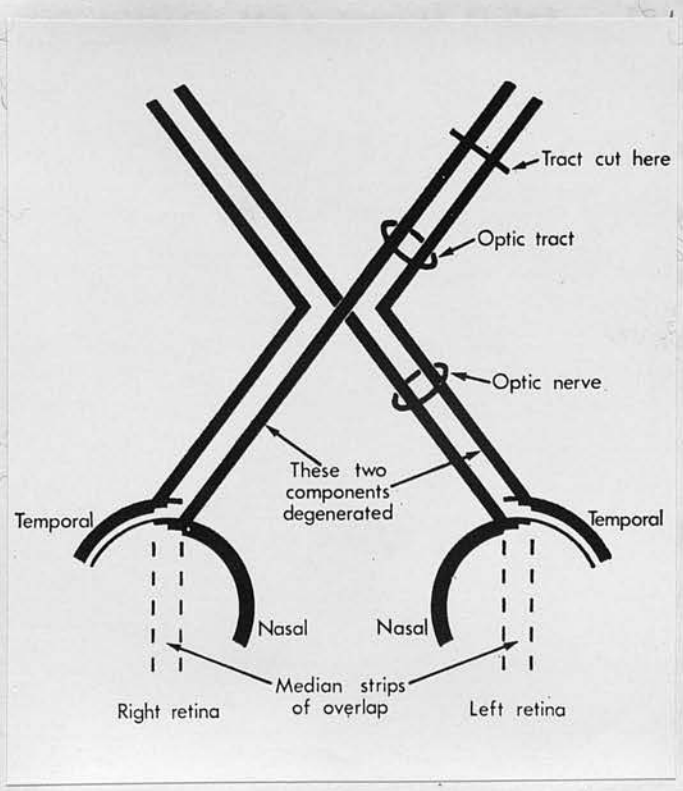


Figure 1
A schematic diagram reproduced from STONE (1966) of the portions of the retina which project to ipsi- and contralateral tracts drawn separately for each retina. The variations in the thickness of the retina indicate the variations in the percentage of ganglion cells which belong to ipsi- and contralateral projecting components.

Behind the upper half of the retina lies the light-reflective tapetum. The lower border of the tapetum runs horizontally just beneath the optic disc,BISHOP P.O., KOZAK & VAKKUR (1962), STONE (1965). By reflecting light back to the receptors it may enhance vision at low levels of illumination.

2. ELECTRICAL ACTIVITY

KUFFLER (1953) recorded from single cat retinal ganglion cells. Small spots of light were focused around the region of the microelectrode tip to study the receptive field of the ganglion cell. This is the area of the retina which must be illuminated to influence the discharge of the cell. Under low levels of illumination there was a spontaneous background activity which was reduced by illumination and anaesthetics. Illumination of the centre of the field of some units produced a discharge during the period of illumination. A light on the annular periphery, on the other hand, only produced a discharge when the light was switched off. Furthermore there was mutual inhibition, that is, a light in the centre reduced the 'Off' response (to a peripheral light), and also a light in the periphery diminished the 'On' response (to a central light). Illumination of the junction between the centre and the surround produced a discharge at 'on' and 'off'. The ganglion cell described is an 'On' centre unit. 'Off' centre units with the converse arrangement were also present.According to WIESEL (1960), at least in the area centralis, 'Off' centre units were found in equal numbers to the 'On' centre units. He recorded from ganglion cells using a micropipette electrode with a tip diameter of less than 0·5μ; this is considerably less than Kuffler's. He showed that the size of the centres of the fields varied from 0·125 mm to 2 mm on the retina (0·5° to 8° of visual angle). This has been confirmed by RODIECK & STONE (1965b) who point out that the receptive field centre of these

small cells would cover an area containing 60 ganglion cells. The observed acuity in a behavioural situation (0.45' and 1.1' for two cats SMITH 1936) is a good deal less than the diameter of the centre of a ganglion cell receptive field.

WIESEL (1960) found that the total diameter of most fields measured was about 2mm, on the retina, but within this the size of the centre varied from field to field. All centres were equally sensitive but there was relatively more peripheral suppression with the small centre cells.

RODIECK & STONE (1965a & b) have presented detailed information about the composition and interaction of centre and peripheral parts of the receptive fields and how the cell reacts to a moving stimulus.

Using elaborate stimulating and recording techniques STONE & FABIAN (1965) and SPINELLI (1966) have described units which do not conform to the classical pattern. Some are 'on/off' units, some have no surround and others are grossly assymetrical to the point of being edge detectors. SPINELLI & WEINGARTEN (1966) observed some directionally sensitive units and also confirmed the observation of BARLOW & LEVICK (1965) that the firing level of the great majority of units was unaffected by the absolute level of illumination. Some exceptions were found though.

So far these studies have been concerned with the light adapted eye. BARLOW, FITZHUGH & KUFFLER (1957a & b) studied the changes that occurred during dark adaptation. This process took up to 3 hours to complete. The drop in threshold occurred in two stages. The first stage represented dark adaptation of the cones and the second stage that of the rods. 'On' and 'Off' centre units had similar thresholds and behaved similarly during adaptation.

When dark adapted they found that the threshold continued to fall as the diameter of the illuminated spot was increased up to 1 mm,

beyond this there was little change. In the light adapted state the threshold again fell as the spot was increased to 1 mm but with further increase to 2 mm the threshold increased due to peripheral inhibition. They concluded that the inhibitory surround was lost during dark adaptation. The loss of periphery was not coincident with the Purkinje shift and the area-threshold curves ran parallel to each other when red and blue stimuli were used under photopic and scotopic conditions. Therefore the change in organization can not be due to a change in rod and cone connections.

The way that the anatomical components combine to produce ganglion cells with the described receptive fields remains a mystery in spite of a recent Golgi study of cat ganglion and bipolar cells by BROWN & MAJOR (1966). The diameter of the dendritic arborizations of a ganglion cell and two bipolar cells is only sufficient to account for the retinal diameter of the centres of receptive fields. GALLEGO & CRUZ (1965) have described associational neurones in the ganglion cell layer of the dog which extend 0.5 - 6 mm. If this is confirmed in the cat, the cell might be concerned in linking the requisite large area to the ganglion cells.

C. THE OPTIC NERVE AND TRACT

An estimate of the number of nerve fibres available for the transmission of visual information from the eye can be obtained by direct counts. Furthermore if each ganglion cell supplies one optic nerve fibre, then a count of ganglion cells should equal the fibre count.

Total fibre counts

BREUSCH & AREY (1942) estimated 117,000 myelinated nerve fibres with an osmic acid stain. With a silver stain, which would impregnate all axons, 119,000 were found. This difference is attributable to experimental error. If any unmyelinated fibres exist they must form

less than 2% of the total number of fibres, although fine fibres below the resolving power of the optical microscope are still possible. Other workers give these estimates : BISHOP, P.O., JEREMY & LANCE (1953): 120,000; van CREVEL & VERHAART (1963) : 64,500; CHILDS : 76,000 (quoted in STONE, 1965). These figures can be compared with estimates of the total number of ganglion cells. BISHOP, P.O. (1953) : 125,000 and STONE (1965) : 90,000. It should be remembered that the differentiation of a ganglion cell from a glial cell is not easy, according to Stone, 10% are not classifiable, and this may account for some of the discrepancies.

Conduction velocity

Different groups of workers have studied the fibre composition of the optic nerve in terms of fibre size and conduction velocity : BISHOP, P.O., JEREMY & LANCE (1953), BISHOP, G.H. & CLARE (1955), CHANG (1956), LENNOX (1958) and van CREVEL & VERHAART (1963). The discrepancies between workers may merely reflect differences in recording and stimulating techniques. It must be difficult to measure such short distances and latencies accurately. The histological sampling technique of BISHOP, G.H. & CLARE (1955) was inadequate. van CREVEL & VERHAART (1963) have discussed sampling difficulties, while at the same time using an incorrect method. There is general agreement that the range of fibre size is from 0.5μ - 11μ with the majority of fibres less than 3μ. More fine fibres cross in the chiasm than enter the ipsilateral optic tract.

The chiasm

It is generally agreed that in the cat about two thirds of the optic fibres cross in the chiasm but it is doubtful if this number has been estimated accurately, POLYAK (1957), HAYHOW (1959) and LATIES & SPRAGUE (1966). STONE (1965) estimates that this temporal area from which the uncrossed fibres arise measures 220 mm^2 and the nasal area 510 mm^2. This would suggest that 70% of fibres cross. HAYHOW (1959) points out that a few fibres decussate on the anterior wall of the tuber cinereum.

It is interesting that there is a species variation in the proportion

of optic nerve fibres which cross in the chiasm. The proportion of uncrossed fibres varies with the degree of frontality of the eyes (DUKE-ELDER, 1958). In man, half cross, in the monkey 2/3, in the rat 9/10 and in the goldfish and pigeon there is complete crossing.

Centrifugal fibres to the retina

The possibility that some control is exerted by the brain over the flow of impulses from the retina has been considered for some time. There is evidence for this in the pigeon (COWAN & POWELL, 1963). There is a massive centrifugal projection to the rabbit olfactory bulb and some evidence for centrifugal fibres to the rabbit retina demonstrable by Glees and Nauta methods (CRAGG, 1962).

Recently BROOKE, DOWNER & POWELL (1965) with the electron microscope, have shown degenerating fibres and terminals in the retina of the monkey and cat after section of the optic nerve, but these might be collaterals of the centripetal fibres. BRINDLEY & HAMASAKI (1966) were unable to find evidence for centrifugal fibres in the cat with the Nauta method. The conclude "...that either the optic nerve contains no centrifugal fibres detectable by silver staining and light microscopy, or if there are such fibres, they are much less susceptible to prograde (Wallerian) degeneration and much more susceptible to retrograde degeneration than most of the centripetal fibres."

SPINELLI & WEINGARTEN (1966) and WEINGARTEN & SPINELLI (1966) have recorded from single units in the curarised and atropinised cat optic nerve, which responded to clicks or electric shocks to the paw but not to light. These auditory or somatic stimuli resulted in changes in the dimensions of retinal receptive fields.

As BRINDLEY (1960) points out in his review of the earlier work, the function of these fibres is obscure. A means of specifically blocking these centrifugal fibres would help if we are to learn more about them.

D. THE DISTRIBUTION OF THE OPTIC TRACT

1. ANATOMICAL METHODS

Recently LATIES & SPRAGUE (1966) have re-investigated the projection of the optic nerve fibres using the Nauta technique. They find five projections: dorsal lateral geniculate nucleus (LGN), ventral lateral geniculate nucleus, nucleus of the optic tract (pretectum), superior colliculus and accessory optic nuclei.

The projection to the dorsal lateral geniculate will be dealt with later.

They judged that the superior colliculus receives 80 - 90% of its fibres from the contralateral hemi-retina.

The nucleus of the optic tract receives 60 - 70% of its fibres after they cross in the chiasm. Some of these fibres come from the contralateral temporal hemi-retina.

The accessory optic nuclei recieve a completely crossed projection a few fibres of which come ffom the temporal hemi-retinae. These two projections would account for the temporal cells which STONE (1966) found remaining after optic tract section. There is no evidence that they project to the LGN.

The results of earlier work have largely been confirmed. The omissions in the work of BARRIS, INGRAM & RANSOM (1935) are no doubt due to their use of the Marchi stain. More recent workers have used the Nauta method. ALTMAN (1962) and SINGLETON & PEELE (1965) have described the projection to the superior colliculus and pretectum. HAYHOW (1959) has given a full description of the accessory optic tract. The degeneration in the ventral part of the lateral geniculate nucleus was thought to be preterminal by HAYHOW (1958) and SINGLETON & PEELE (1965) although ALTMAN (1962) thought they were only fibres of passage. Degeneration has not been reported in any other part of the thalamus, hypothalamus or mid-brain. Although ALTMAN (1962) has said "In limited regions of

the pulvinar, adjacent to the lateral geniculate body, some indications of preterminal arborizations were observed. However, extensive areas of the pulvinar were entirely free of degenerated preterminal fibers.", the other groups of workers have not agreed. The importance of this region will be discussed on p. 46.

BARRIS et al. (1935) observed collaterals of both small and large fibres passing into the dorsal nucleus of the LGN, the main fibre passing by the brachium to the superior colliculus. GLEES (1941) stated that the majority of crossed optic fibres in the LGN were collaterals which left the optic tract at right angles, the main fibres proceeding to the tectum. O'LEARY (1940) showed that all large fibres bifurcate at the dorsal nucleus entry zone. Thick branches arched immediately into the dorsal nucleus; the thin branches could not be traced further, it is conceivable that they continued to the superior colliculus.

It would be interesting to compare collicular visual receptive fields with geniculate ones to see if they deal with the same population of ganglion cells or if they are concerned with different attributes of the visual world.

2. ELECTROPHYSIOLOGICAL METHODS

BISHOP, G.H. & CLARE (1955) suggest that there are four groups of fibres within the tract. Each group has a characteristic conduction velocity. The fastest activating predominantly the A and A1 laminae of the LGN. The next group relays through lamina B of the LGN to the thalamus medial to the LGN. The third group runs to the pretectum and the fourth, slowest, group activate the superior colliculus.

The evidence is not conclusive. The estimates of conduction velocities depend on the accurate measurement of latencies (see p. 9 for criticisms). Conduction velocity could not be measured for the third and fourth groups as the presynaptic responses could not be

recorded. Instead an estimate was made from the post-synaptic latency and the duration of the synaptic delay was assumed. More reliable evidence for grouping comes from the threshold differences for electrical stimulation. The grouping was not found to be exclusive, some of the group I fibres were found running to the superior colliculus. Although four peaks in frequency distribution histogram of the optic nerve fibres could not be found they did confirm qualitatively that fibres of appropriate size went to the appropriate end-station.

The optic tract input to the pretectum and superior colliculus has been described. These regions are not directly related to the experimental work which will be presented and therefore they will not be discussed further.

THE LATERAL GENICULATE NUCLEUS

1. General Anatomy

This will be considered in some detail as in the experiments to be described, lesions were made in the LGN and adjacent regions to trace the projections to the cortex. An attempt was also made to see if the lamina B had a different function to the A lamina.

THUMA (1928) described the trilaminate appearance of the dorsal lateral geniculate nucleus (LGN) terming the layers pars dorsalis A, pars dorsalis A_1 and pars dorsalis B. In parasagittal section the laminae form an S-shaped structure with the pars dorsalis A lying most dorsal, the pars dorsalis B the most ventral and pars dorsalis A_1 lying in between. For brevity, it is proposed to follow HAYHOW (1958) and refer just to lamina A, lamina A_1 or lamina B.

The dorsal concavity of the A lamina embraces an antero-superiorily directed hilum containing the origin of the optic radiation. The laminae A and A_1 are separated by a conspicuous interlaminar fibre plexus which contains scattered large cells. The cellular appearance of the two laminae is similar, with a uniform distribution of cells (10 - 40μ in diameter) found in numbers inversely related to their size.

The interlaminar fibre plexus between A_1 and B is less marked than between A and A_1 and is more of a cellular transition zone characterized by the presence of scattered, large deeply staining cells, the central interlaminar nucleus (HAYHOW, 1958). The lamina B cells differ from the A and A_1 cells. They are spindle shaped cells 20 - 25μ long. The optic tract is split by the pars ventralis to form lateral and medial rami. The former sweeps over the posterior surface of the dorsal LGN and the latter enters the nucleus ventral to the B lamina.

The anterior perigeniculate nucleus is an attenuated irregular layer of small cells forming a cap over the anterior and dorsal

Figure 2

A semi-schematic drawing of a parasagittal and a cross section through the lateral geniculate nucleus of the cat. a) correspond approximately to the Horsley-Clarke planes of reference, two of which are indicated by the vertical and horizontal arrows. The approximate plane of the parasagittal section is indicated by the vertical arrows in (b). The latter represents a cross section through the nucleus along a plane approximately indicated by the diagonal arrows in (a), the plane of section being about 45° to the Horsley-Clarke vertical. The ventral LGN has been inserted in (a) although it would really be found in a more lateral plane (from HAYHOW, 1958).

Figure 3

A dorsal view of the LGN (left side) of the cat, showing the representation of the visual field (see Fig. 27 for a standard perimeter chart). The solid line 180° represents, the horizontal meridian, the 250° line is in the lower visual field. Dotted lines represent angles of deviation from the centre of the field,(from SENEVIRATNE, 1962).

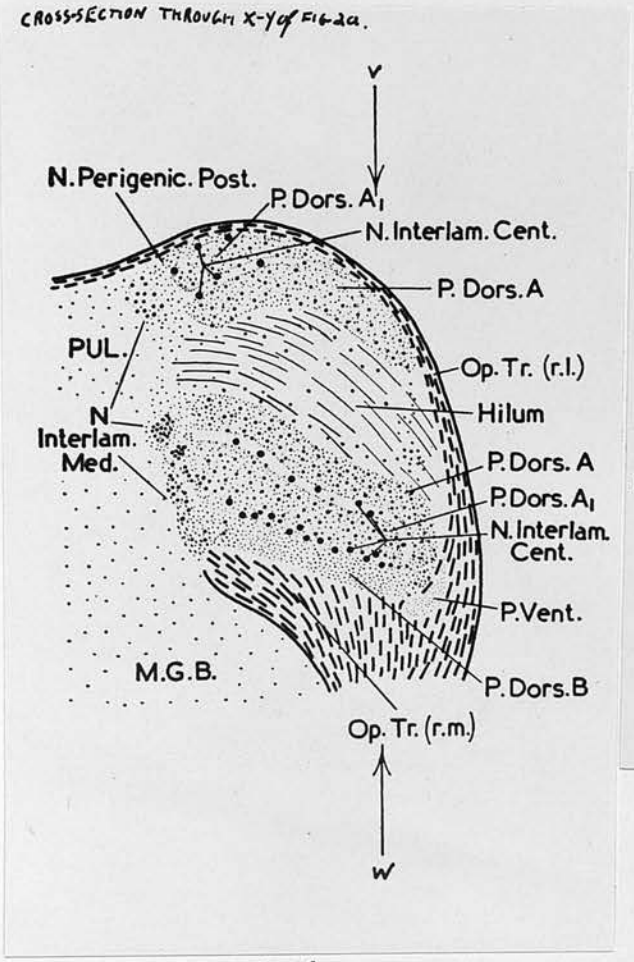


FIG 2b.

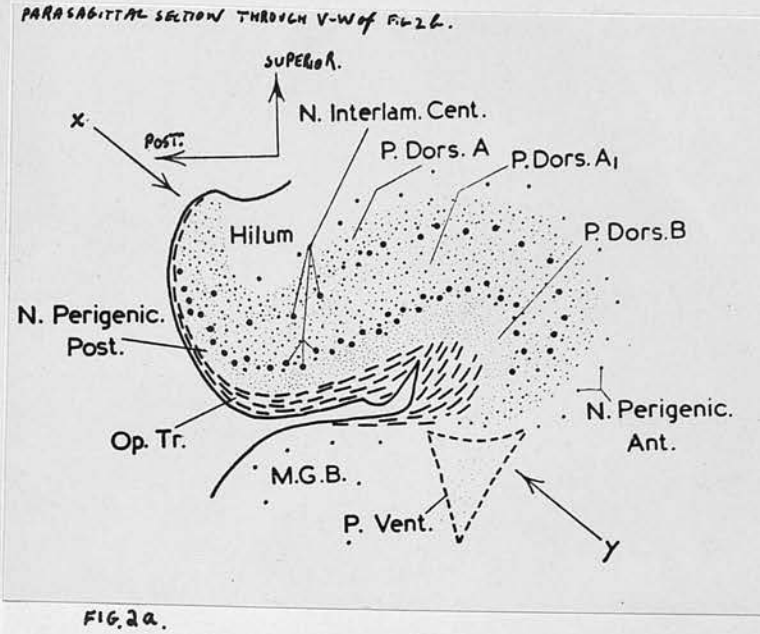


FIG 2a.

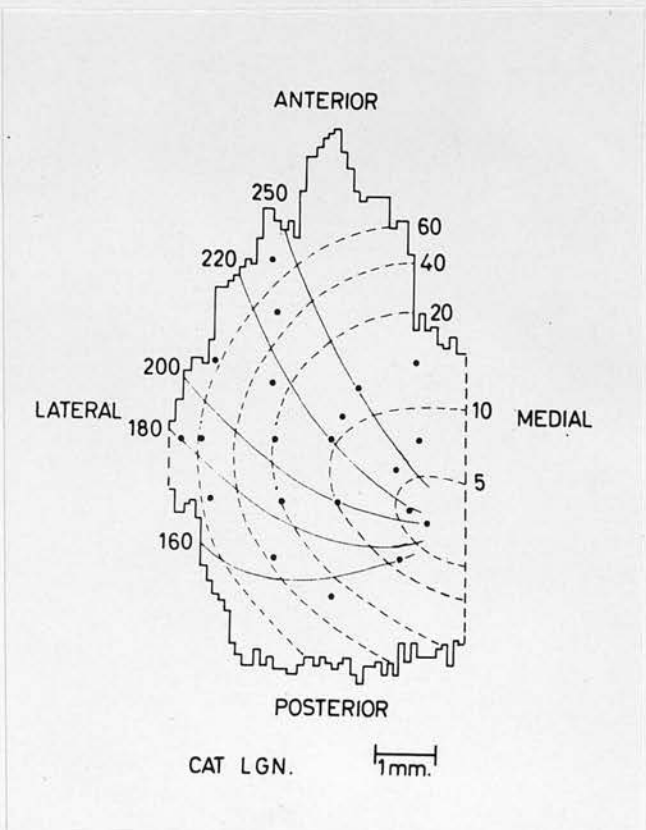


FIG. 3.

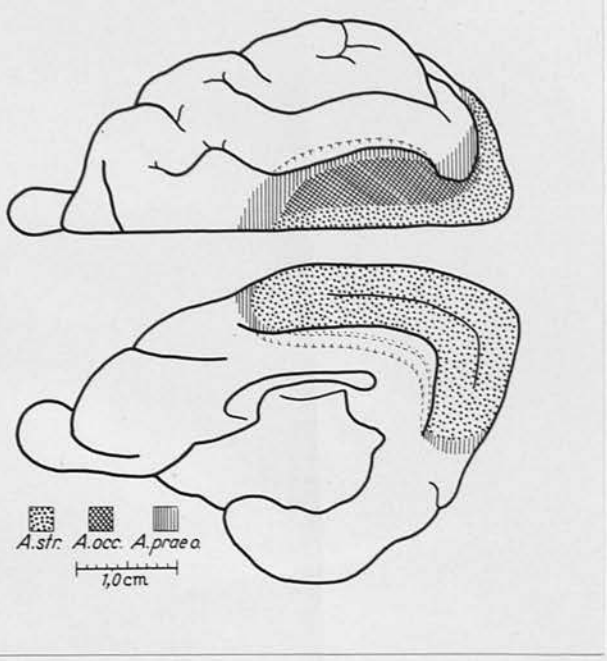


FIG. 4.

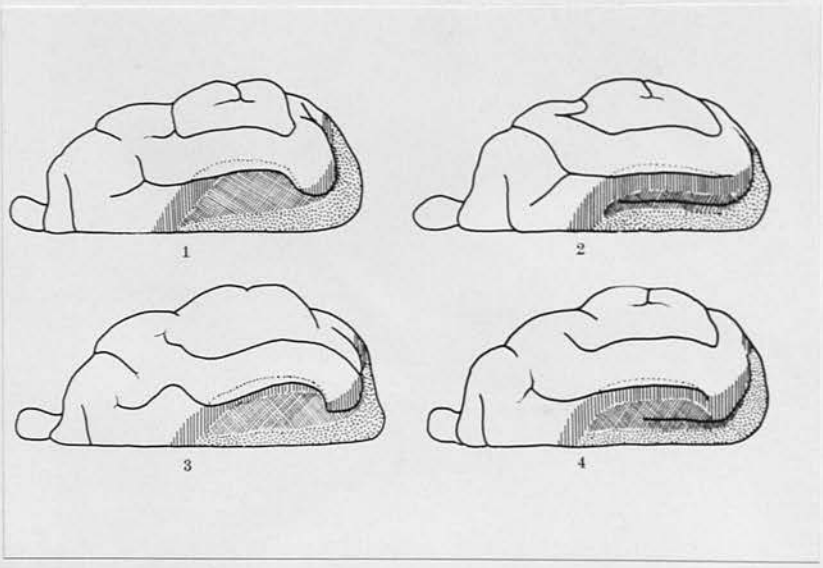


FIG. 5.

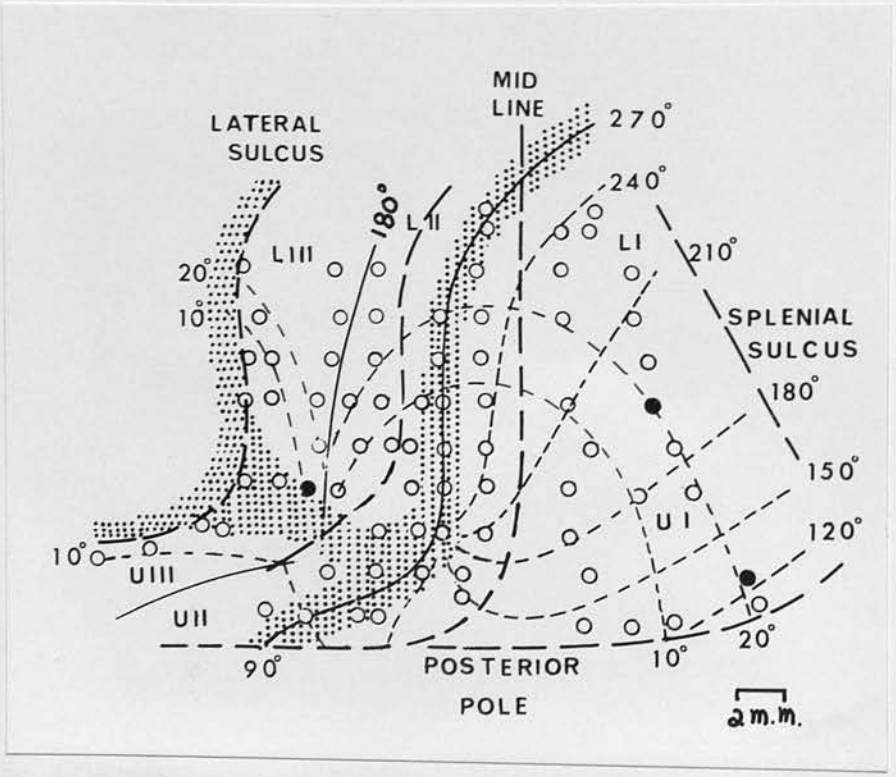


FIG. 6.

Figure 4

A dorsal and a medial view of the cat brain showing the visual cytoarchitectonic areas, (From OTSUKA & HASSLER, 1962)

Figure 5

Dorsal views of cat brains showing the visual cytoarchitectonic areas and variations in gyral pattern. Note the intralateral accessory gyrus in 2. (From OTSUKA & HASSLER, 1962)

Figure 6

The representation of the visual field on the cat cortex (WHITTERIDGE, personal communication). The brain is represented flat (medio-laterally) by taking measurements around the perimeter of the lateral gyrus from the mid-line. The dotted lines refer to the angles of the visual field (see perimeter chart, Fig 27) The dotted bands emphasise the position of the vertical meridian and the area centralis. UI, UII, UIII refer to the upper quadrants and LI, LII, LIII to the lower quadrants of the field in Visual I, II, and III.

o Points used for the construction of the map.

• Points with a discrepancy of more than 3^{o} from the expec expected position.

surfaces of the lamina A.

The posterior pergeniculate nucleus is a cap of small cells over the posterior end of the lamina A_1 and is in apparent continuity with lamina B.

The medial interlaminar nucleus is a cluster of cells similar to those in the main part of the nucleus. It lies ventro-medial to the pars dorsalis and merges with the pulvinar medially. This description has been added to by HAYHOW (1958), STONE & HANSEN (1966) and LATIES & SPRAGUE (1966) who have depicted the boundaries of the nucleus. But it must be remembered that they are not likely to be as sharp as the diagrams suggest. There is some evidence for a vertical trilamination within the nucleus.

The pars ventralis is a small pyramidal cellular mass lying between the medial and lateral rami of the optic tract, ventro-lateral to the pars dorsalis and separated from it by a thin fibre stratum containing a few cells similar to those found in lamina B.

2. The distribution of optic nerve fibres within the LGN

HAYHOW (1958) has reviewed the earlier work and with the Nauta method has presented conclusive evidence that the ipsilateral fibres end in the A lamina and the contralateral fibres in the A and B lamina. The intralaminar arborizations are highly specific in this respect. In the interlaminar zones the optic tract fibres form tangentially orientated plexuses. An overlap between the crossed and uncrossed optic preterminal fibres occurs here. Interlaminar cells may have a dual innervation.

STONE & HANSEN (1966) have described the pattern of preterminal Nauta degeneration seen in the LGN after small retinal lesions. The true size of the lesions was checked by examining whole retinal mounts of the damaged eyes to determine the position and total area

of degenerating ganglion cells.

A topographical organization between the retina and the LGN was found. This was in accord with the electrophysiological maps of BISHOP, KOZAK, LEVICK & VAKKUR (1962) and SENEVIRATNE & WHITTERIDGE (1962). Fig. 3. Areas within the region of overlap at the area centralis (STONE, 1966) projected to both LGNs.

The projections of adjacent areas of the retina overlap extensively in the LGN. This overlap is greatest for peripheral areas of the retina, the central areas having a more discreet projection.

For the pericentral regions the distribution of fibres to the three laminae described by HAYHOW (1958) was confirmed. No contralateral degeneration was found after a temporal retinal lesion. Therefore the ganglion cells which remain in the temporal retina after cutting an optic tract do not project to the LGN (see Fig. 1) their destination is a mystery, (but see p. 16 for suggestions).

The retinal lesions in two cats were less than 0.4 mm diameter and were accurately located in the centre of the area centralis. Preterminal degeneration was seen in the ipsilateral lamina A_1 and the contralateral A lamina. This was not described by LATIES & SPRAGUE (1966) who made rather larger retinal lesions. It is suggested that the B lamina does not receive a projection from the centre of the retina.

A projection to the medial interlaminar nucleus was described, but not from the two central lesions. Degeneration from the temporal retina to the ipsilateral medial interlaminar nucleus tended to be centrally placed. HAYHOW (1958) with unilateral enucleations showed that this nucleus was organised in three vertical columns, the central column being supplied by the ipsilateral eye. A contralateral projection to either one, but not both of the outer columns was found by STONE & HANSEN (1966) and LATIES & SPRAGUE (1966).

All agree that the projections overlap. The superior retina projects to the anterior part of the medial interlaminar nucleus and conversely, the inferior retina to the posterior part (as with the main body of the LGN). STONE & HANSEN (1966) did not find any organization in any other direction, whereas LATIES & SPRAGUE describe their central lesions which were rather larger than the STONE & HANSEN ones, projecting to the nucleus close to the interlaminar centralis nucleus while more peripheral lesions were inferolaterally situated, lying under the medial part of lamina B.

Further work is required to settle the dispute about the projection from the area centralis to the medial interlaminar nucleus and the lamina B. This is of importance because if these connections do not exist then it is unlikely that these two parts of the LGN are concerned in acute vision.

It is not known if one optic nerve divides to supply the contralateral A and B laminae and also perhaps the medial interlaminar nucleus or if each of these structures is supplied by a separate optic nerve. Since the temporo-nasal distribution of ganglion cells is uniform (STONE, 1965) it would lead to the surprising results in the latter case that the density of cells projecting to the A lamina is less than to the A_1 lamina.

HAYHOW (1958) also studied the terminal degeneration using Bielschowsky, Cajal and Glees stains. With these methods there was no precise correlation with the cytoarchitecture of the nucleus. He accounts for this by the difficulty of identifying true degenerating boutons in sufficient numbers in cat (HAYHOW, 1959). The boutons of the normal cat LGN vary considerably both in morphology and total number revealed. These variations appear to be related principally to the depth of impregnation of the sections. The only absolutely reliable criterion of a degenerating optic bouton is the attachment to it of a degenerating preterminal fibre (HAYHOW, 1958)

FILLENZ (1961) believes that HAYHOW'S survival time of 5 - 7 days was too long. Using only 3 days she found clear evidence of degenerating boutons in the interlaminar plexus from both eyes. She did not comment on the similarity between normal and degenerating boutons.

The difficulty of differentiating normal from degenerated boutons casts doubt on the estimate of GLEES (1941) that one principal cell of the cat LGN makes forty synaptic contacts and that one optic fibre is distributed to an area covering about ten cells. Furthermore no details or photographs are given on which his calculations were based. ~~Electron microscopy results to be discussed are also contrary to this view.~~

As the fibres enter their layer of termination they bifurcate repeatedly, the terminal branches retaining a predominantly parallel orientation directed towards the upper surface of the lamina. This was likened by TELLO (1904) to the arborization of a cypress tree. His Golgi figure shows this pattern in the 'B' lamina although O'LEARY (1940) and HAYHOW (1958) disagree on this point. The arborizations of an optic tract fibre are restricted to a particular lamina and the adjacent intralaminar region. However, within a lamina the terminal arborizations of different tract fibres partially overlap. Each geniculate cell is likely to be in contact with several different optic tract fibres (O'LEARY, 1940).

In his Golgi studies of kitten LGN O'LEARY (1940) classified geniculate cells as principal cells with axons extending into the optic radiation and short axon cells with axons confined to the lamina in which the cell body lies. The axonal arborizations of the latter cells may surround the cell or be eccentric. The basket type ending is common, enclosing the bodies of principal cells. The axons of some cells in the B layer are reported to enter the optic tract and could not be traced further. He thought that they might either re-enter the nucleus or pass medially.

The dendrites of principal and short axon cells are chiefly intralaminar in distribution, although marginal cells exist, the dendrites of which pass along the interlaminar strata.

3. Electron microscopy

PETERS & PALAY (1966) in a detailed study of laminae A and A1 of the cat LGN have largely confirmed the earlier findings of SZENTAGOTHAI (1963). Their most important conclusions are that the majority of axons terminate on the small dendrites and relatively few on the cell bodies. They describe synapses as occuring in glomeruli which are partially encapsulated by astrocytic processes. The appearance of Golgi stained material has been fancifully described; 'grape-like' dendritic terminations are seized by 'claw-like' endings of axons. This description receives some support from the electron microscopy which shows a second type of axon terminal which is thicker and centrally placed in the glomerulus. Each preterminal fibre breaks up into a number of claws which contribute to several glomeruli and the central axon enters into more than one glomerulus. In this way one glomerulus may receive from more than one optic tract fibre. Furthermore, within the glomerulus the central axon forms axo-axonal contacts on the peripheral axons, the central axon terminal possessing vesicles. Axo-axonal contacts are thought to be inhibitory, see ECCLES (1964). These contacts have also been described by SZENTAGOTHAI (1963) and presynaptic inhibition at the LGN has been observed by ANGEL, MAGNI & STRATA (1965). This means then, that both types of terminals may excite the dendrites but at the same time the central axon may inhibit the peripheral ones.

One may speculate that the few axosomatic synapses also may be inhibitory since this seems to be the case elsewhere in the

nervous system (see ECCLES, 1964). These endings may well be derived from the short axon cells with basket type endings described by O'LEARY (1940). This is supported by SZENTAGOTHAI'S (1963) comment that the axosomatic synapses persist after bilateral enucleation.

It will be of interest to discover the origin of the two types of axon terminals as one appears to be inhibitory to the other. Are these inhibitory fibres non-optic afferents or are both fibres optic afferents and this an anatomical manifestation of the increased suppression found by HUBEL & WIESEL (1961) when they compared the receptive fields of optic tract fibres and LGN cells?

4. Other afferents

It seems fairly certain that other afferents influence the activity of the LGN but the details are not adequately established. The first piece of evidence is the persistence of some terminals after bilateral enucleation (SZENTAGOTHAI, 1963).

Further evidence is that after a gross lesion which destroyed the midbrain at the intercollicular level, 85% of geniculate units were silenced by retinal pressure block compared with 61% in the intact animal, suggesting that there is some influence from the midbrain (LEVICK & WILLIAMS, 1964).

Electrical stimulation of the midbrain and pontine reticular formation affects the evoked response at the LGN (HERNANDEZ-PEON 1961; SUZUKI & TAIRI 1961; OKUDA 1962; HOTTA & KAMEDA 1964), but the effect of current spread to other structures of fibres of passage has not been considered. This is obviously important since NAUTA & KUYPERS (1958) and SZENTAGOTHAI (1963) were unable to trace Nauta degeneration from reticular stereotaxic lesions to the LGN,

although the possibility of a polysynaptic pathway remains.

Stimulation of the ipsilateral lateral gyrus (KWAK, 1964) or the posterior end of the middle suprasylvian gyrus and the nearby portion of the lateral gyrus influences the response of the LGN to a test optic tract electric shock or photic stimulation (WIDEN & AJMONE MARSAN, 1960; AJMONE MARSAN &MORILLO, 1961).

The function of these afferents to the LGN is not known, although HERNANDEZ-PEON (1961) suggests that they may be concerned with visual attention.

5. Efferents from the LGN

The chief projection, the optic radiation, will be discussed fully under striate cortex.

If all the axons of the LGN terminate only in the visual cortex then all neurones should show retrograde reaction after its removal. This is the case in the monkey (POLYAK, 1933) and the rat (LASHLEY, 1934). In the cat there is conflict. MINKOWSKI (1913) found unchanged neurones remaining in the LGN after lesions of the lateral gyrus. SMITH (1937) reported "Complete absence of ganglion cells" after removal of the lateral and mid-suprasylvian gyri bilaterally. This was confirmed by DOTY (1958) who found that removal of the lateral gyrus only was not sufficient. On the other hand, BADEN, URBAITIS & MEIKLE (1965) found some neurones persisting in the LGN after bilateral removal of lateral and middle suprasylvian gyri and PEACOCK & COMBS (1965) found normal neurones remaining in the LGN after hemi-decortication. They did not comment on their laminar distribution. FISCHMAN & MEIKLE (1965) after removal of the area 17 and the lateral and posterolateral gyri and superior colliculi found medium sized neurones remaining in laminae A and A1 of the LGN. They remarked on the absence of large neurones.

One might expect three classes of neurones to remain after removal of the striate cortex:

1. The short axon cells described by O'LEARY (1940).
2. Any neurone whose essential projection is thalamic.
3. Neurones with an essential projection to extra-striate cortex.

Although there has been some conflict, evidence is mounting that normal neurones do persist in the LGN after even extensive removals of cortex. This supports the idea of short axon cells and/or a subcortical projection. The short axon cells could have been mistaken for imperfectly stained principal cells and as will be shown, the evidence for a subcortical projection from the LGN is not altogether convincing. Therefore additional support is welcome, although it is unfortunate that the two possibilities have not been differentiated. Persistence of cells in the LGN after hemidecortication and a knife cut medial to the LGN would be good evidence for the short axon cells. This was attempted. It is curious that in contrast to the cat, degeneration is complete in the monkey and the rat.

There is some evidence for a projection from the LGN to the rest of the thalamus and midbrain. RIOCH (1931) observed fibres running between the principal laminae (A and A1) of the LGN and the pulvinar and between the laminae parvocellular and magnocellular (lamina B) to the pulvinar pars posterior and the nucleus posterior. This is based on normal Weigert material which gives no indication of direction and does not distinguish from fibres of passage.

BARRIS et al. (1935) were unable to establish details of a projection from the LGN to adjacent medial structures as stereotaxic lesions of the LGN damaged cortico-fugal fibres which ran through the LGN to these more medial regions. Nevertheless, degeneration from the LGN lesions in two areas, nucleus lateralis

pars posterior and the dorsal half of the stratum opticum of the superior colliculus was not adequately explained by damage to fibres of passage.

ALTMAN (1962) also made stereotaxic lesions of the LGN and followed the degeneration with the Nauta method. Unfortunately he failed to take into account the electrode track, the cortical lesion and the cortico-fugal fibres mentioned by BARRIS et al. (1935) and the retino-collicular and retino-pretectal fibres (LATIES & SPRAGUE, 1966). The lesions were large and other structures were damaged (pars ventralis, pulvinar, adjacent forebrain structures). The ventral lesions damaged the optic tract and this would explain some of the degeneration in the superiorcolliculus. Therefore it would be unwise to lay too much emphasis on his poitive finding that the LGN projects not only to the striate cortex but also to the pretectum, superior colliculus, pulvinar, pars ventralis of the LGN, posterior nucleus, postero-lateral nucleus and the suprageniculate nucleus. His negative findings are of course valid.

BISHOP, G.H. & CLARE (1955) have recorded post-synaptic responses to stimulation of the optic nerve in the region just medial to the LGN. A synapse in the B lamina is assumed, because the medial thalamic response is first evident when the stimulus threshold is raised to that of their group III fibres which they had shown ran to the B lamina. As the stimulus strength is increased, the responses in the B lamina and the thalamus increase and are both maximal at the same stimulus intensity. However, they remark on the inconspicuousness of the response and wondered if the fibres were diffused over a wide area or, as seems likely, that this is not the sole projection of the B lamina. After all, if the lamina B only projected to the medial thalamus then one would expect it to be intact after removal of the visual cortex. This appears unlikely from previous

work. As there is some uncertainty this was investigated.

BUSER, BORENSTEIN & BRUNER (1959) have reported evoked potentials to light in this region medial to the LGN using gross electrodes and BRUNER (1965) has recorded from single units in this region. The responses cannot be due to a direct input from the optic tract (p.17). A connection from the LGN seems the most likely, although other sources e.g. superior colliculus or pretectum cannot be excluded. If it is confirmed that the LGN is the source of the projection then which laminae are concerned? Is it just the B lamina as BISHOP , G.H. & CLARE (1955) suggest?

Definite knowledge of input to this region is desirable, because it will be shown that it projects to the middle suprasylvian gyrus and other cortical areas which may be activated by stimulation within other modalities. Furthermore, the lateral part of the middle suprasylvian gyrus may be a site of complex pattern analysis and it may even be comparable to the infero-temporal region of the monkey. (See Discussion).

6. Electrophysiology

The termination of the ipsilateral optic tract fibres in lamina A1 and the contalateral fibres in the A and B laminae has been confirmed by COHN (1956), HUBEL & WIESEL (1961), BISHOP, P.O. et al. (1962[b]) and SENEVIRATNE (1962).

Binocular interaction is uncommon. Thus, BISHOP, P.O., BURKE & DAVIS (1959) found that less than 8.5% of geniculate cells responded to electrical stimulation of either optic nerve. Indirect interaction was noted more frequently. This could be seen when either nerve would fire the cell after a long latency, or when stimulation of only one optic nerve caused the cell to fire but the rate could be influenced by the other optic nerve. This has been confirmed by ERULKAR & FILLENZ (1960) and FILLENZ (1961), who used

diffuse flashes of light. Niether group of workers accurately localised the site of their binocular responses. BISHOP, P.O. et al. (1962[b]) show one unit which responded to a 2° visual stimulus to either eye. The cell was situated in the A/A1 junction. SENEVIRATNE (1962) found some units in the lamina magnocellularis, the large cells at the A1/B junction, which could be activated by stimulation of either eye. In one instance this was confirmed by an electrolytic marking lesion at the recording site. HUBEL & WIESEL (1961) were unable to find binocularily activated units.

SENEVIRATNE (1962) and SENEVIRATNE & WHITTERIDGE (1962) have produced maps of the retinotopic localization on the lateral geniculate body. This method has the advantage of producing a more detailed map than can be obtained by the anatomical methods of tracking degeneration from retinal lesions. They recorded from single units with fine tungsten electrodes while stimulating one or both eyes with small areas of light. Central vision was found on the medial edge of the body and the lateral field was represented in the lateral part of the body; lower visual field was anterior and upper field, posterior. The lower visual field, particularly between 180° and 240° meridians had a greater representation than the upper field. A disproportionately large area was devoted to central vision compared with the peripheral visual field. (See Fig. 3).

There was some evidence of a diminutive mirror image or second visual area in the medial interlaminar nucleus. It was difficult to record from this area because of its small size. (However, these results have been confirmed and extended by VESBAESYA & WHITTERIDGE, personal communication).

A segregation of 'on centre' and 'off centre' units was found within the laminae. The former were found predominantly in the upper halves and the latter in the lower halves of the A and A1 laminae.

Units in the B lamina behaved somewhat differently. There were more 'off centre' units and these tended to have a continuous discharge in the dark which could be depressed with barbiturates. There was no segregation of 'off centre' from 'on centre' units within the lamina. They tended to have larger receptive fields with low thresholds to light. It is interesting that SMITH et al. (1966) found the greatest horizontal spread of optic tract axons in lamina B. The size of the field of some of the 'off centre' units depended on the size and duration of the preceding stimulus, the effect wearing off within a few minutes. There was less peripheral inhibition and HUBEL & WIESEL (1961) commented that these cells had longer latencies and reponded at lower frequencies than in other laminae.

Some of these results of SENEVIRATNE have also been reported by BISHOP, P.O. et al. (1962 a & b). These authors particularly discuss many of the basic problems inherent in the idea of topographical localization in the visual system.

HUBEL & WIESEL (1961) were more concerned with the organization of the receptive fields of individual units in the lateral geniculate body. Units with receptive fields in the projection area of the area centralis had smaller field centres and showed stronger peripheral suppression of their centres than cells with fields in more peripheral regions. This would be advantageous for acute vision.

In some units, almost all the geniculate cell spikes were preceded by potentials due to impulses in a single optic tract fibre. In a few units there were several distinct sizes of optic tract potentials, suggesting for these units the convergence of a number of excitatory tract fibres on a single cell. The long duration of the potentials, the presence of summation and their ability to trigger the geniculate cell suggested that they were of presynaptic

origin. In simultaneous recordings of geniculate cells and optic tract fibres it was observed that there was an increase in peripheral suppression at the geniculate level. The optic tract and the geniculate cells are not related in a strictly one to one manner that might be expected at a relay station. The response to a moving spot of light was independent of the direction of movement.

These experiments tell us that topographic localization is still maintained at the LGN and furthermore, there is a miniature second visual representation of unknown function. There is further evidence that the A and A1 cells differ from the B cells although this may be obscured to some extent by some degree of binocular overlap in the interlaminar plexuses. HUBEL & WIESEL tell us that the LGN is not merely a relay station as there are slight differences between the receptive fields of optic tract and LGN units. The number of laminae in the LGN differs in the various species; the rat LGN is unlaminated whereas in primates there are six laminae. The reason for lamination in the LGN remains a matter for conjecture. Although some differences between the laminae have been observed, in the cat the B lamina may not receive an input from the area centralis and there is some evidence that it projects medially into the thalamus. The cells in the different laminae have been shown to have different properties (SENEVIRATNE, 1962, in the cat; de VALOIS JONES 1961, and JACOBS 1966, in the monkey).

CEREBRAL CORTEX

1. Cytoarchitectonics

Although the striate area of monkey and man is immediately obvious , as will be shown, these and other areas in the cat are not. Consequently, one should be sceptical of those who claim that they can make many subdivisions with exact boundaries. The cytoarchitectonic features change markedly with the changing pattern of gyri and sulci, complicating the appearance of an 'area' which crosses a sulcus. The influence of subjective bias is likely to be great and it is unfortunate that none of the workers has adopted a procedure to circumvent this. In the cat, WINKLER & POTTER (1914) have presented a series of cross-sections marked with their cytoarchitectonic boundaries. The discrepancies between adjacent sections are alarming.

Dissatisfied with this map and earlier ones by BRODMAN (1906), CAMPBELL (1905) and GUREWITSCH & CHATSCHATURIAN (1928), OTSUKA & HASSLER (1962) have studied the problem again (Fig. 4). They produce no evidence for either the reliability or validity of their boundaries. However, their area striata or area 17 corresponds fairly closely to the visual area I described using electrophysiological means (TALBOT & MARSHALL, 1941 and BILGE, SENEVIRATNE & WHITTERIDGE, 1963). It is by no means as clearly defined as in the monkey. Adjacent to it on the dorsal surface of the brain is the area occipitalis (area 18). The area prae-occipitalis (area 19) forms a complete ring around these areas, and the ring is completed in the s. splenialis inferiorly. The boundaries of these areas are even less well defined. This may mean that the functional areas are not sharply divided.

Variations in sulcal pattern were noted. This principally affects area 18 which has a smaller surface lateral extent when it

is infolded into the intralateral accessory sulcus, which they describe (Fig.5 - 2 & 4).

The maps produced by these different workers all differ, not only in the nomenclature of the areas, but also in their boundaries. This no doubt reflects the difficulty of demarcating these areas.

Few non-cytoarchitectonic workers have related their finding to these areas, although their terminology may suggest otherwise. To obviate this difficulty, reference will usually be made to the gyri. When cytoarchitectonic numbers are used in accordance with recent workers, they will refer to the areas defined by OTSUKA & HASSLER (1962) unless otherwise stated. This is for descriptive convenience and does not imply acceptance of their findings.

2. Golgi studies

The appearance using the Golgi stain, of the visual cortex both between the hemispheres and on top of the lateral gyrus has been studied in detail by O'LEARY (1941).

SHOLL (1953, 1955) has made an important advance by giving quantitative details. Unfortunately he does not give the location of the part of the visual cortex which he used. It seems probable that his calculations are based upon the striate cortex defined by O'LEARY (1941) which roughly corresponds to Visual I described by the physiologists. It would be interesting to know if he would have found differences between parts of cortex concerned with peripheral and central vision, although CHOW, BLUM & BLUM (1950) and COWEY (1964), did not find any significant difference in the cell density of monkey. But SOLNITZKY & HARMAN (1946) found that the cortex was thicker in the macular area than in the monkey's more peripheral cortex.

SHOLL attempted to validate his sampling by showing that (with

one exception) in each layer the neuronal density, expressed as a ratio of the neuronal density of the layer containing the fewest cells, was the same whether the neurones had been counted in a Nissl or a Golgi preparation. H e attributed the discrepancy in the deepest layer to difficulties in demarcating the grey-white boundary and distinguishing between neurone and glia with the Nissl stain.

Three groups of afferents were distinguished:-

1. Coarse afferents which terminate around Gennari's line. He believed these to be the radiation fibres from the LGN, but there is no conclusive evidence for this (p.39). The branches of one fibre extend for 650μ (i.e. covering a circle of this diameter) in a direction parallel to the surface of the cortex. One afferent fibre is likely to influence 0.1 mm^3 of cortex containing 5,000 neurones. However, these figures must be kept in perspective. One fibre is only influencing about 3 sq. mm of cortex which is only a small proportion of the whole striate cortex. This then, is in no way incompatible with topographical localization.

2. Afferents which end in the region between the line of Gennari and layer 1. These are thinner, thought to be of commisural origin, and extend covering a circle of 150μ diameter.

3. This group ramifies in the outermost layer of the cortex and in part at least are the terminals of recurrent collaterals from cells in deeper layers. This group of afferents may extend for several millimetres.

The dendrites of stellate cells and the basal dendrites of the pyramidal cells extend 250 - 500μ around the perikaryon (In this paper shrinkage factor was not calculated so the distance will be even greater then reported). Within this volume there are 2,000 - 4,000 perikarya. Details are given of the frequency of branching

of dendrites. The dendritic density falls exponentially with distance from the cell body. He estimated that 75,000 fibres/ mm^2 leave the visual cortex compared with 25,000 afferent fibres/ mm^2. Thus we now have a measure of the area and volume of cortex into which a single optic radiation fibre extends and the maximum number of neurones which might be directly influenced by this fibre has been calculated. CRAGG (1966) has estimated the synaptic density (by counting boutons on electron micrographs) as 6 x 10^{11}/cc and the neuronal density (Nissl stain and light microscopy) as 1 x 10^8/cc in the monkey visual cortex. Dividing one figure by the other he concludes that average number of synapses per neurone is 6,000.

3. Afferents to the cortex

a. Lateral and postero-lateral gyri.

(i) Anatomical investigations - Retrograde Degeneration

Early retrograde degeneration studies were made by Monakow (1885) and Minkowski (1913) are reviewed by WALLER & BARRIS (1937) who have provided further information. In five cats lesions were restricted to the lateral gyrus but not to any one cytoarchitectonic area within this gyrus. Retrograde degeneration was found only in the LGN, its distribution is in keeping with the distribution of the visual fields as later presented by the physiologists.

DOTY (1958) showed that removal of the middle third of the lateral gyrus (area 18) with slight penetration of the white matter produced very little degeneration in the LGN. This degeneration was attributed to the damage to the radiation. GAREY (1965) reports retrograde degeneration in the medial interlaminar nucleus of the LGN from a lesion placed lateral to area 17.

Anterograde Degeneration

POLYAK (1927) is frequently quoted as having shown the cortical

distribution of the optic radiation. However, this rests on one animal (Expt. 3) in which a large cortical lesion also penetrates the optic radiation. The evidence that these radiation fibres were damaged is only indirect, they may not have been completely damaged, other fibres were almost certainly involved and finally the Marchi technique may have failed to show all the fibres.

A more direct approach is to make a lesion in the LGN and study the cortical degeneration. ALTMAN (1962) has done this but has only commented that Nauta degeneration was found in the striate cortex without giving further details.

GLICKSTEIN, MILLER & SMITH (1964) found an area of Nauta degeneration roughly corresponding to Visual II of TALBOT (1942), after a stereotaxic lesion of the contralateral LGN. This is the only report of a contralateral projection from the LGN via the corpus callosum.

In view of the unsatisfactory state of our knowledge on these points and the surprising finding of GLICKSTEIN et al. (1964). Projections from the LGN were investigated in the manner just suggested.

(ii) Electrophysiological Investigations

TALBOT & MARSHALL (1941) recorded with gross electrodes the cortical responses of greatest amplitude and shortest latency to a 2^{o} flash in the cat anaesthetized with nembutal. Each hemi-cortex received a projection from the contralateral half fields of both eyes. A map of the projection of the visual field (Visual I) on the cortex was made.

TALBOT (1942), described a second visual area (Visual II) lateral to the vertical meridian, oppositely disposed and confined anteriorly to the lateral gyrus and posteriorly to the suprasylvian gyrus. The Visual I corresponds approximately to Brodmann's area 17 and Visual II to his area 18. The response in Visual II was

identical to the response from Visual I for the latency of the primary wave and the latency and amplitude of the 'off' wave, although DOTY (1958) and BILGE, SENEVIRATNE, & WHITTERIDGE (1963) state the amplitude here is greater than more medially. It was not depressed by 'narcosis or cautery' of Visual I and it fired independently of the application of convulsants or the local stimulation of Visual I. This suggests that Visual II has an independent input from the LGN. In one cat DOTY (1958) showed that three weeks post-operatively photically evoked potentials could be recorded from a strip of mid-lateral gyrus, medial to which, striate cortex had been removed. This suggests that Visual II has an input independent of the striate cortex.

BILGE, SENEVIRATNE & WHITTERIDGE (1963) have recorded from single units or small groups of units with tungsten electrodes and have confirmed TALBOT'& MARSHALL'S (1941) description of Visual I. In general their Visual I co-incides with the striate area of OTSUKA & HASSLER (1962). They have also shown that the lower visual field has a greater area of representation than the upper and confirm Talbot and Marshall that the central visual areas have a greater cortical representation than the peripheral visual field.

In a study of the visual areas lateral to Visual I, WHITTERIDGE (personal communication, see Fig. 6) agrees with TALBOT (1942) that the medial boundary of Visual II is the vertical meridian (i.e. Visual I and II meet along a line representing the vertical meridian). However, Whitteridge disagrees with Talbot over the lateral boundary of Visual II. Whitteridge's evidence is that the horizontal meridian in Visual II is split and it is this which forms the lateral boundary of Visual II. This boundary is also the medial border of Visual III which is a mirror-image of Visual II.

HUBEL & WIESEL (1965) imply that Visual II and III meet at the

peripheral field. However, an examination of their figures shows that the receptive fields also approach the horizontal meridian at this boundary. In fairness it must be pointed out that their technique of examining a small area of cortex is not suited to determining the relationship of the Visual II/Visual III boundary to the visual field. The Whitteridge map (Fig. 6) explains why they were sometimes unable to find much representation of the peripheral field when they examined the Visual II/Visual III border near the area centralis. Furthermore, from a lesion close to the vertical meridian in Visual I, they show the Nauta degeneration (Fig. 8) immediately lateral to the Visual II/Visual III boundary. This is not comprehensible if this boundary is thought to represent the peripheral visual field, but it is explicable in terms of the Whitteridge map for a lesion near the horizontal meridian. The exact site of the lesion is not given. HUBEL & WIESEL (1965) have also attempted to combine anatomical and physiological observations in the same animal. In general this relates Visual I to area 17, Visual II to area 18 and Visual III to area 19. It is doubtful if the cytoarchitectonic boundaries can be localized sufficiently accurately and their Fig. 34 (see Fig. 8) suggests subjective bias (see p.56).

Examination of WHITTERIDGE'S map (Fig. 6) shows that in Visual III central vision is disproportionately represented compared with Visual I, at the expense of peripheral vision.

DOTY (1958) failed to confirm topographical retino-cortical relations. This would have serious implications for our concepts of visual function. However, his work is open to a number of objections. He did not routinely fix the eye or check that it was vertical, so that their movement during the experiment can not be excluded. He centred his perimeter about the blind spot, com-

plicating the interpretation of the results unnecessarily. He recorded from two points 4 mm anterior to the position of the horizontal meridian as later determined by BILGE et al. (1963) and instead of locating the best position for evoking potentials of the shortest latency, he measured the amplitude and latency for different horizontal positions of the light. It is not surprising that he did not find the horizontal meridian. DOTY (1961) accepted that the topography could be demonstrated.

Summary

Three topographically organised cortical visual areas have been described. Retrograde degeneration studies indicate that Visual I receives a direct projection from the LGN. There is a little evidence that Visual II has a projection independent of Visual I but on the whole our knowledge is far from satisfactory and the existence of a second visual area in the LGN only adds to the confusion - does this project to the visual cortex and if so, to which visual area? Therefore these problems have been investigated.

b. Middle suprasylvian gyrus

(i) Anatomical evidence

In the work of WALLER & BARRIS (1937) no lesions were restricted to the suprasylvian gyrus. Posterior lesions of the middle suprasylvian and lateral gyri not only produced retrograde degeneration in the LGN but also in the pulvinar. More anterior lesions produced degeneration in the rostral LGN and the posterior part of the lateral nucleus (which probably correspond to nucleus lateralis posterior of more modern terminology). One lesion affected the anterior part of the middle suprasylvian and the middle of the middle ectosylvian gyri, the lateral gyrus was spared. No degeneration was found in the LGN but it was present in the posterior and intermediate parts

of the lateral nucleus.

MARSHALL, TALBOT & ADES (1943) report no degeneration in the LGN after circumscribed lesions in the suprasylvian gyrus. No other details were published.

Retrograde degeneration in the pulvinar, lateralis posterior, the lateral corner of n. lateralis dorsalis anterior and probably the nucleus posterior from bilateral middle-suprasylvian gyri lesions have been described by WARREN, WARREN & AKERT (1961) and HARA (1962). They report some degeneration in the LGN but this is attributed to the lesion penetrating the radiation. This is confirmed by BADEN, URBAITIS & MEIKLE (1965) who in addition, removed the lateral gyrus bilaterally.

(ii) Physiological evidence

Photically evoked potentials may be recorded on the middle suprasylvian gyrus but the pathway is disputed. It is conceivable that the input comes a) directly from the LGN, b) from some other thalamic nucleus, c) from the lateral gyrus, or d) from some other part of cortex which is visually excitable. A combination is possible. No group of workers has convincingly established their case. Fig. 7 is a summary of their different views which will now be discussed in detail as the middle suprasylvian may have important visual functions.

MARSHALL, TALBOT & ADES (1943) reported photically evoked potentials of two types on the middle suprasylvian gyrus. One type is described as having considerably amplitude and a long latency. It can be influenced by the application of Nembutal or picrotoxin to the lateral gyrus. Therefore they ascribed this response to fibres from the lateral gyrus to the middle suprasylvian gyrus. It persists after both striate cortices have been removed and the tectal region separated from the geniculo-thalamic region by a sagittal knife cut.

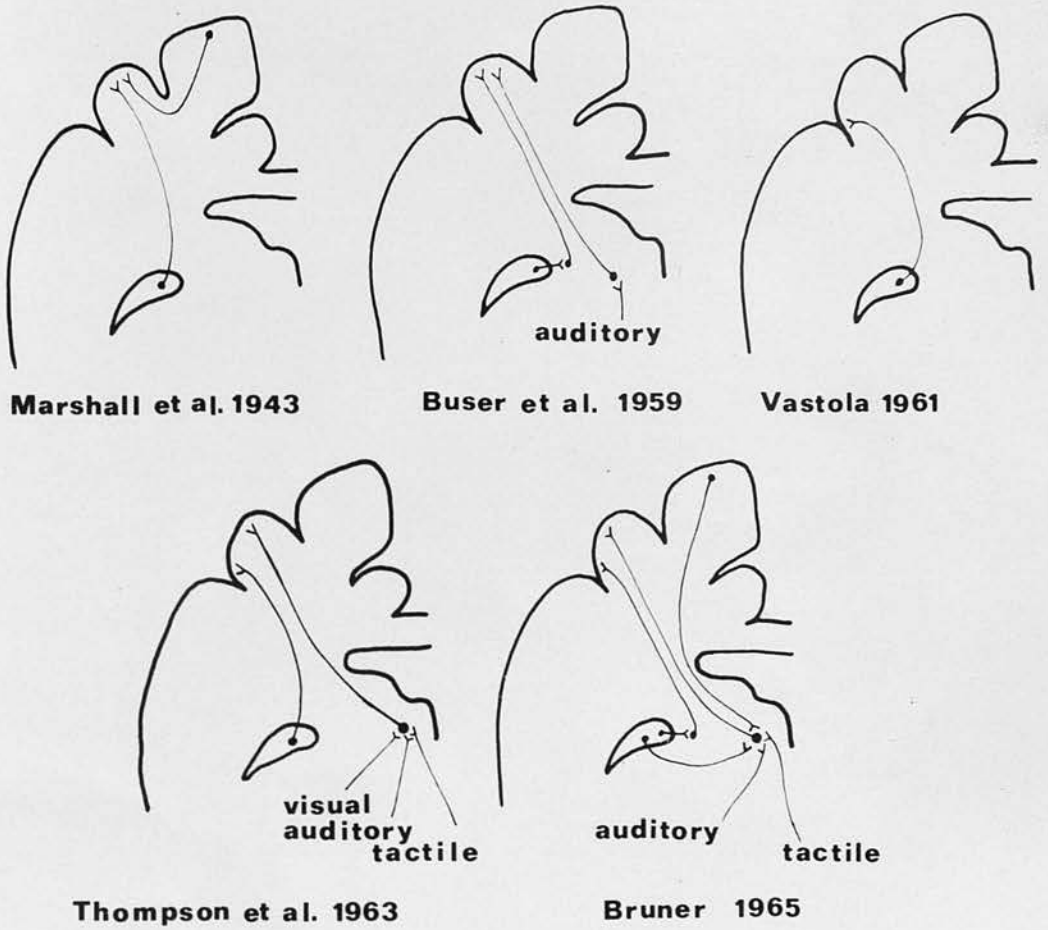


Figure 7

Summary diagram of the various projections proposed to the middle suprasylvian gyrus.

Immediately after the striate cortex has been removed the second type of response is still found. After sufficient time has elapsed for retrograde degeneration to be well developed in the LGN the response is no longer found in the middle suprasylvian gyrus. Since it is unlikely that the same animalswas used for the two sets of recordings it is unfortunate that the histology is not presented to prove that the removal was adequate. If this were satisfactory, an interesting possibility remains, that the fibres project to the middle suprasylvian gyrus are collaterals of the main fibres to the striate cortex and are insufficient to 'sustain' the LGN neurones after striate removal.

BUSER & BORENSTEIN (1959) in the unanaesthetized, curarised cat and BUSER, BORENSTEIN & BRUNER (1959) in the cat anaesthetized with chloralose describe "association areas" on the surface of the middle suprasylvian gyrus and on the anterior part of the lateral gyrus where visual tactile and auditory responses could be elicited. The lateral gyrus response could be abolished by bilateral removal of the gyrus or modified by thetopical application of potassium chloride, Nembutal or strychnine without affecting the response in the association area, but this interference would be unlikely to influence the medial wall of the hemisphere.

It is unlikely that the projection originates from the mesencephalic reticular system since extensive destruction of this area did not abolish the response.

The thalamus medial to the LGN was postulated as a relay station. Responses to flashes of light could be recorded from the posterior nucleus, dorsal part of the lateral posterior nucleus and the inferior part of the pulvinar. Responses to clicks could be found in the inferior part of the lateral posterior nucleus and the suprageniculate nucleus. There was some overlap between these areas. This is not a recording artefact as BRUNER (1965) has found single

units in this region activated by flash. Some units were polysensory. Electrical stimulation of these nuclei produced responses on the middle suprasylvian gyrus but not in the striate regions. VASTOLA (1961) points out that Buser's stimulating electrode was within 1 mm of the MGB and could have activated the auditory cortex which was then recorded in the nearby suprasylvian area. This is unlikely however as many of the points of stimulation in Buser et al.'s Fig. 14 were well away from the MGB and more important in Fig. 13 (presumably the one criticised by Vastola) responses were absent over the auditory area of the cortex.

Electrical stimulation of the LGN evoked a response in the suprasylvian gyrus. This is not good evidence implicating the LGN in this pathway as the current might easily have spread to other structures e.g. optic tract or medial thalamus.

VASTOLA (1961) proposed a direct projection from the LGN to the lateral wall of the suprasylvian gyrus (SL). Responses could be picked up here by a bipolar electrode after an electric shock had been given to the contralateral optic nerve. This was not abolished by extensive undercutting of the lateral gyrus and medial wall of the hemisphere. Lesions separating the LGN from the medial thalamic structures and the pretectal area had no effect, but anodal polarization of the ipsilateral LGN abolished the response.

He also concluded that both the striate and the suprasylvian responses originate in the geniculate cells innervated by the group of fastest conducting fibres in the optic tract and that a last group of fibres then project from the LGN to the striate region and a slower group project to both striate and suprasylvian areas. These results must be accepted with caution since they depend upon small differentes in latencies and estimates of distance.

THOMPSON, JOHNSON & HOOPES (1963) and THOMPSON, SMITH & BLISS (1963)

describe four areas - anterior and posterior suprasylvian, anterior lateral and peri-cruciate gyri. Stimuli delivered to the peripheral neurones of the auditory, somatic or visual system converge upon single neurones in the subcortex which project to these four areas.

The chief evidence for this statement concerns the unresponsive period of the cortex when a response was elicited after applying a stimulus in one modality for a short duration, a second stimulus was incapable of evoking a response. The duration of this unresponsive period was the same whether the two stimuli were within the same or different modalities.

The distribution on the cortex of the response to stimuli within each modality was the same, This conflicts with BUSER et al. (1959). However, it seems unlikely that with gross recording electrodes that the boundaries can be drawn sufficiently accurately to be certain one way or the other. The waveform and the latency of the response in these areas were similar but differed from the lateral gyrus and the SL response. The amplitudes of the responses in the four areas showed similar variation over a period of time. The response amplitudes in the lateral gyrus and SL varied in a similar manner - this adds support to VASTOLA'S (1961) claim for a direct projection from the LGN to SL.

The thalamo-cortical organization of the system is unknown.

Against their interpretation are the findings of DUBNER & RUTLEDGE (1964, 1965) and DUBNER (1966) who recorded from single units in the anterior lateral and middle suprasylvian gyri. They showed that the convergence of sensory input upon neurones activated by flash, click or contralateral forepaw electrical stimulation is unequal. Furthermore, in the middle suprasylvian gyrus more units responded to flash then click, whereas the converse was true for the anterior lateral gyrus. However, this preferential responsive-

ness was dependent on the stimulus used (electrical shock or natural) and the amount of the chloralose anaesthetic. This argues strongly against the single undifferentiated 'association' system of THOMPSON et al. (1963).

BRUNER (1965) also distinguishes between the medial part of the suprasylvian gyrus (SM) and the lateral part (SL). The SL response in contrast to SM is independent of the visual projection to the lateral gyrus as the response is not affected by the removal of the lateral gyrus. In contrast, SM fatigues readily, and is depressed by the application of potassium chloride to the lateral gyrus. The anterior limbic, the pericruciate region and SM are similar in their behaviour and are activated by auditory and somatic stimuli. The photic response of SL but not the lateral gyrus is much diminished by the injection of potassium chloride into the lateral posterior nucleus suggesting that it is a relay station for SL. A direct projection from the LGN to SL can not be excluded with certainty as the response is not completely abolished.

The postero-median nuclei (centre median and the adjacent nuclei) are postulated as the relay to SM. Responses in these nuclei can be evoked by visual, auditory and bilateral somatic stimuli. In jection of potassium chloride depresses but does not abolish the evoked potentials in SM, SL is unchanged. No direct evidence is offered as to how the visual impulses arrive at these nuclei or their path to the cortex.

In accord with the anatomical findings, a short latency response was recorded in SL to electrical stimulation of the lateral gyrus, (this could be blocked by a knife cut between the gyri), and responses could also be found in lateralis posterior, pulvinar and postero-median nuclei. The first two projections confirm BERESFORD (1961) who used Nauta. Inspite of this and in contrast to SM, the visual response at SL was not modified by stimulation, depression

by topical potassium chloride, or the removal of the lateral gyrus. This projection has not been included in the Figure.7.

The response in SM to electrical stimulation of the lateral gyus is not abolished by a knife cut between the gyri but it is abolished by the injection of potassium chloride into the posterior part of the ventro-median nucleus. The application of strychnine or potassium chloride to the lateral gyrus modifies the response in the medial thalamus for visual but not other stimuli. It is suggested that the lateral gyrus has a specific modifying action through postero-median nuclei upon the visual impulses received at SM. A projection from the lateral gyrus to lateralis posterior has been described by Beresford using the Nauta method.

BIGNALL, IMBERT & BUSER (1966) confirm that photically evoked responses can be found on the dorsal surface of the lateral suprasylvian, anterior lateral, anterior sigmoid gyri and also note activity in the dorsal part of the ectosylvian and orbital gyri. These responses persist after removal of the contralateral cortex and extensive thalamic ablation medial to the LGN. As this ablation was not complete it cannot be inferred that activity in these areas is due either to a direct projection from the LGN or from the striate cortex.

Conclusion

A direct pathway between the lateral gyrus and SL has been demonstrated by BRUNER (1965) and this has been confirmed by HUBEL & WIESEL (1965) using the Nauta method (see p.53). But BRUNER (1965) and VASTOLA (1961) have shown that this is not an 'essential' connection, there being some other visual input. Connections between the lateral gyrus and SM are indirect (BRUNER, 1965), and this is confirmed by the absence of Nauta degeneration here after a lesion of area 17 (HUBEL & WIESEL, 1965, see p.53).

VASTOLA (1961) has shown that the SL response is not dependent

on the thalamus medial to the LGN. BIGNALL et al. (1966) have shown that the middle suprasylvian gyrus response is not dependent on the postero-median group of nuclei. However, BRUNER (1965) and BUSER et al. (1959) have shown that these thalamic regions make a substantial contribution to the suprasylvian response. As the electro-physiological observations are not entirely in agreement and as some of the conclusions are questionable, further anatomical studies would be desirable.

It would be interesting to know how these sites interact and affect the response to a more natural stimulus than a shock to the optic nerve or a flash to the whole eye. A study of the receptive fields of individual units would also be useful. There is reason to believe that these units might have highly complex organization. (See p.137).

IPSILATERAL CORTICO-CORTICAL CONNECTIONS

(a) From Lateral and Postero-lateral Gyri

A knowledge of these connections is necessary to show how visual information is spread across the cortex from the receiving area in the striate cortex.

(i) Anatomical Evidence

POLYAK (1927) using the Marchi method showed that fibres ran from the posterolateral and lateral gyrus junction to the middle suprasylvian gyrus and a few fibres went to the ectosylvian gyrus.

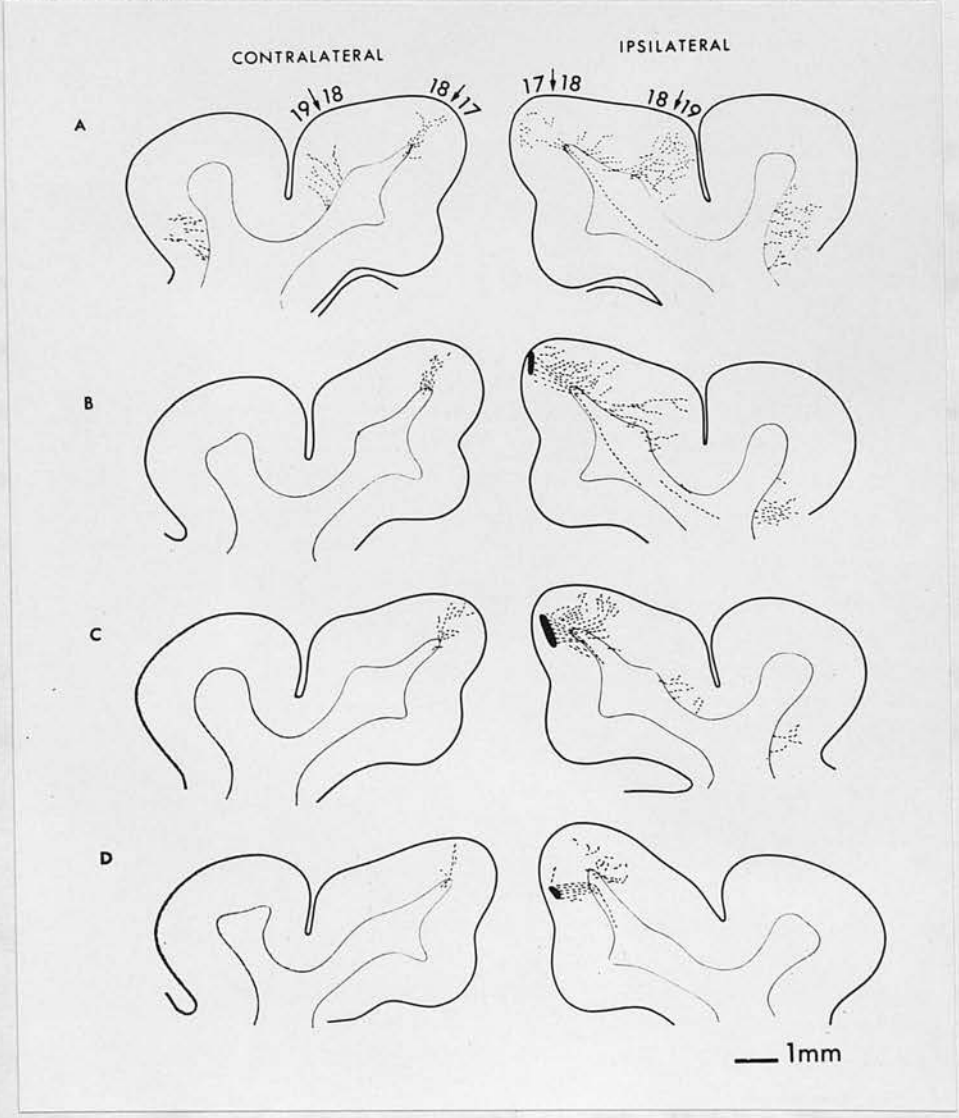


Figure 8

Nauta degeneration from a lesion shown in black. Reproduced from HUBEL & WIESEL (1965a), Fig. 34.

HUBEL & WIESEL (1965) (Fig. 8) have used the Nauta technique to follow degeneration from lesions in a small number of animals which they believe to be in area 17 as defined by OTSUKA & HASSLER (1962). They find degeneration in areas 18 and 19 and the lateral wall of the middle suprasylvian gyrus. Fibres were noted to run directly and not radially into and out of the white matter. From a needle stab degeneration extended only for a few millimetres in an antero-posterior direction. These results confirm some of the findings of BERESFORD (1961), POLLEY & DIRKES (1963) who also used the Nauta method.

(ii) Physiological evidence

A projection from the lateral gyrus to the middle suprasylvian gyrus was suggested with strychnine neuronography by GAROL (1942). This was confirmed by CLARE & BISHOP, G.H. (1954) who demonstrated a point to point localisation. Electrical stimulation of the lateral gyrus produced an evoked potential localised to 2 - 3 mm of middle suprasylvian gyrus, slightly anterior to the point stimulated (See also MARSHALL et al. 1943; BRUNER, 1965).

GAROL (1942) strychninised the lateral gyrus about half way along its length. Responses were found in the anterior lateral gyrus, middle suprasylvian gyrus, anterior suprasylvian gyrus and possibly the orbital gyrus.

These findings have been confirmed and extended to include the dorsal margin of ectosylvian, anterior sylvian sigmoid and orbital gyri by IMBERT, BIGNALL & BUSER (1966). They recorded the gross response evoked by stimulation of mid-lateral gyrus (which they found to be the most effective part) in cats lacking the contralateral cortex, thalamus and sometimes the lateral geniculate body. These responses were interpreted as due to orthodromic conduction since responses could not be evoked in the reverse direction. It

is unlikely that the results were due to intra-griseal current spread which stimulated directly at the recording site, since an inactive region was found between stimulating and recording electrodes. The chance of current spreading to deeper subcortical fibres of passage could not be excluded but was minimised by using a low voltage for stimulation. No check was made that this was so. The pattern of responses over the whole brain varied from animal to animal. This is perhaps not surprising in view of the massive removals which varied in detail from animal to animal. The anaesthetic did not seem to be a factor since similar maps were produced using chloralose or decerebration. Latencies were quite long (up to 10 m.sec) so polysynaptic cortical pathways are possible.

(b) From Suprasylvian Gyrus

(i) Anatomical evidence

The large lesion in POLYAK'S (1927) experiment 2 is predominantly in the middle suprasylvian gyrus but there was also damage to the posterolateral gyrus and to the white matter beneath it. Marchi degeneration products were found over a wide area of lateral, posterelateral, middle suprasylvian, the dorsal posterior suprasylvian, dorsal part of posterior ectosylvian and the middle ectosylvian gyri.

CRAGG (1965) with Nauta has shown that the middle (in an antero-posterior direction) of the middle suprasylvian gyrus projects to both banks of splenial sulcus, the lateral half of the lateral gyrus, and the cingulate gyrus. More anterior or posterior lesions do not project to the splenial sulcus and have only very slight cingulate projections.

(ii) Physiological evidence

The experiments of IMBERT et al. (1966), previously described, showed a connection from the anterior end of the middle suprasylvian

gyrus to the pericruciate gyrus predominantly the anterior sigmoid gyrus.

GAROL (1942) using strychnine showed a projection to the middle and anterior parts of the lateral gyrus and there was a possible connection to the anterior sylvian gyrus.

Summary

There is a little good anatomical evidence for projections from area 17 to 18, 19 and the suprasylvian gyrus and from part of the suprasylvian gyrus to area 18, 19 and the cingulate gyrus, but topographical localization details have not been fully elucidated. There is weaker evidence for a much wider projection using strychnine neuronography and electric shock. The limitations of these methods have been discussed (p.7).

CALLOSAL CONNECTIONS

a) Anatomical evidence

The lesion of Experiment 1 of POLYAK (1927) was not definitely located within area 17. Marchi staining showed "...callosal fibres to the striate area (field 17)", his figure suggests this includes the whole surface of the lateral gyrus and the medial wall of the hemisphere. It was also present in the contralateral suprasylvian gyrus.

HUBEL & WIESEL (1965) (Fig. 8) traced Nauta degeneration from a needle stab in area 17 to the contralateral medial edge of area 18, area 19 and the lateral wall of the suprasylvian sulcus. They say a few fibres to the lateral edge of area 17 could not be excluded, however their Figure 34 reproduced in Fig. 8, shows the contralateral 17/18 boundary placed far more medially than usual (cf. OTSUKA & HASSLER 1962). Suggesting that there is a projection to area 17 from the opposite area 17, POLLEY & DIRKES (1963) also refer to a projection to the contralateral 17.

EBNER & MYERS (1965) report on the distribution of Nauta degeneration after completely cutting the anterior commissure and corpus callosum in one cat and hemi-decorticating another one. No cyto-architectonic investigations were undertaken. The distribution of the degeneration was related to the map published by OTSUKA & HASSLER (1962). No degeneration was reported in area 17 but it was dense in area 18 forming a complete ring around 17. The inferior part contained only moderate degeneration, here it is not recognised as 18 by OTSUKA & HASSLER (1962). Area 19 was degeneration-free except for a strip which runs across it at a level which appears to be about 6 mm anterior to the occipital pole (i,e, roughly corresponding to the level at which the area centralis is represented upon the cortex). Degeneration was found on the middle suprasylvian

gyrus although it was absent from part of its surface: here it was only found in the depths of the bordering sulci. A close comparison of the reproduced cross-sections and the reconstructed dorsal view (Fig. 9) shows that the area of degeneration which they related to area 18 is drawn too laterally. In the posterior sections this error may be due to incorrect alignment of the sections with the vertical (cf. CLARKE & HENDERSON, 1911).

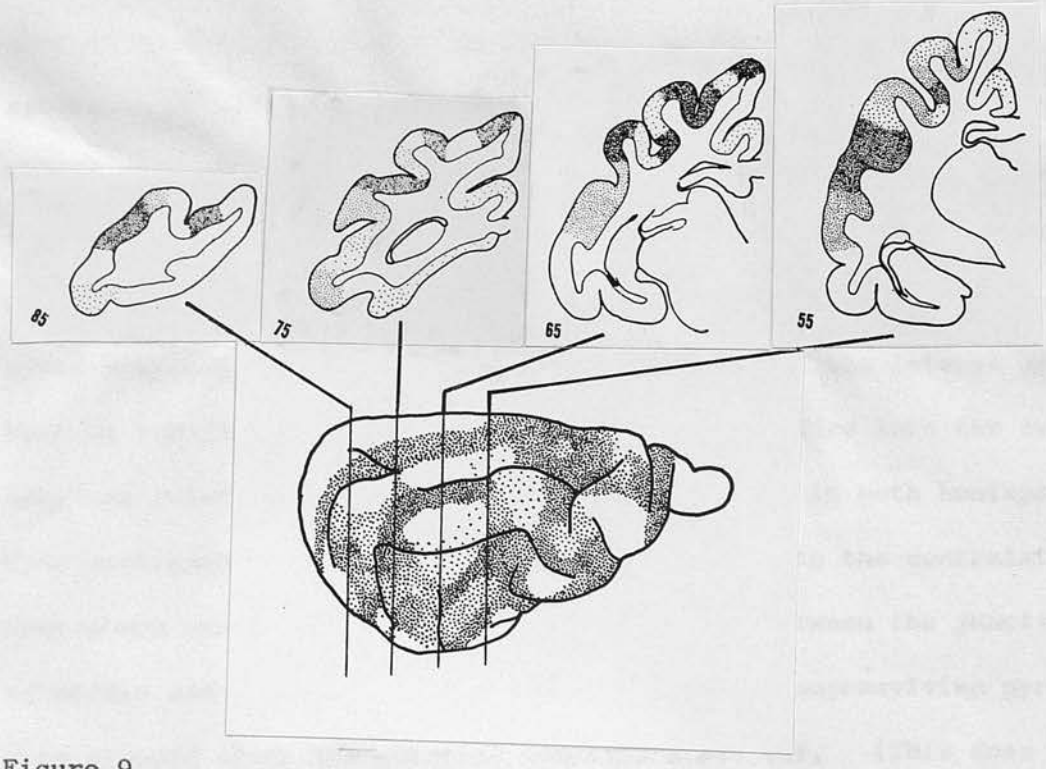


Figure 9

The dorsal view of the right half of a cat brain showing Nauta degeneration following removal of the left half of the brain. The figure has been constructed from cross sections, four of which are reproduced and the appropriate level indicated. Note the discrepancy on the lateral gyrus between the cross-sections and the dorsal view. (From EBNER & MYERS, 1965).

In view of these criticisms it seems that the lateral edge of area 17 does receive transcallosal fibres.

b) Physiological evidence

CURTIS (1940) applied single electric shocks to one hemisphere and recorded the response contralaterally with bipolar silver ball electrodes. Small or no potentials were recorded when symmetrically opposite points of the postero-lateral and medial half of the posterior part of the lateral gyrus were studied. Large potentials were evoked at symmetrical middle suprasylvian, middle ectosylvian and anterior lateral gyri. There was no evidence for transcallosal connections from one area 17 to the other.

GAROL (1942) showed that local strychninization of the lateral edge of the postero-lateral gyrus and the posterior part of the lateral gyrus caused firing to symmetrical points in the opposite hemisphere. No transcallosal firing was evoked by strychninizing the medial part of the postero-lateral gyrus (i.e. area 17). The middle suprasylvian gyrus fired symmetrically and to the anterior and middle parts of the lateral gyrus. The middle part of the lateral gyrus projects to the middle and anterior parts of the lateral gyrus. Part of the posterior suprasylvian gyrus did not fire into the symmetrical point but suppressed electrical activity in both hemispheres. Upon sectioning the corpus callosum all firing into the contralateral hemisphere ceased except for diminished spikes between the junction of middle and posterior ectosylvian and adjacent suprasylvian gyri. This stopped after the anterior commissure was cut. (This does not necessarily prove that the anterior commissure is the route since sectioning it may have caused deterioration in the animals condition at the end of the experiment).

Summary

The early physiological evidence is of little help in determining the areas with callosal connections. Apart from the general criticisms of these methods (see introduction) their maps are of

'coarse grain' and give no information about areas between the hemispheres.

The cross-sections of EBNER & MYERS (1965) are of value in establishing the terminations of callosal fibres. HUBEL & WIESEL (1965) have given information about the beginning and end of the callosal projection from a lesion close to the representation of the vertical meridian in one animal. The relationship of this projection to the area 17/18 boundary has been questioned.

Further anatomical details of these connections are required. Secondly, there is no information as to the physiological nature or possible function of these connections. Experiments were conducted to clarify these points.

CORTICO-FUGAL CONNECTIONS

BERESFORD (1961), (and confirmed by ALTMAN, 1962) has followed the Nauta degeneration from the posterior lateral gyrus and the medial postero-lateral gyrus and has found a similar distribution from these areas but he does not appear to have distinguished between the different cytoarchitectonic areas. Degeneration was found in the ipsilateral superior colliculus, nucleus of the optic tract, posterior nucleus, LGN - layers A and A1 but not B, reticular nucleus of the thalamus, pulvinar, lateralis posterior nucleus, and pons. It was not certain that the fibres ended in the last three regions, they may have been fibres of passage.

BERESFORD (1962) discusses the possibility that the described degeneration is retrograde in nature. No conclusive argument was put forward but it seemed likely that the degeneration was not retrograde.

GAREY (1965) reports a topographic projection from the lateral and postero-lateral gyri onto the superior colliculus.

From the middle part of the middle suprasylvian gyrus CRAGG (1965) has described with the Nauta method, projections to a number of thalamic and mesencephalic nuclei.

FUNCTIONAL ORGANIZATION

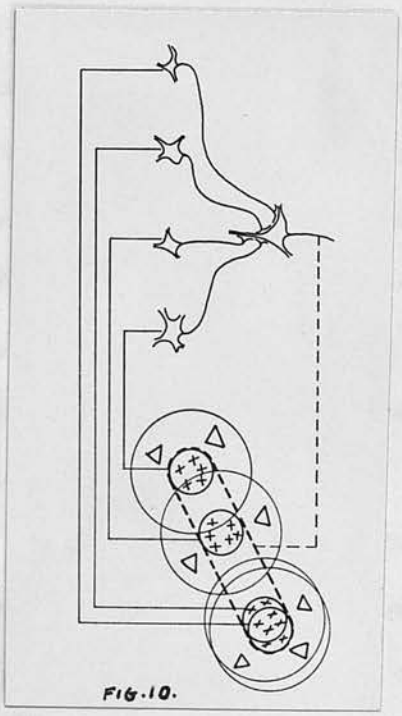
The description given so far of the topographical projections of neurones onto area 17 and the adjacent cortex gives only a static impression of the system. It is a wiring diagram which gives little idea of the function of the parts. This defect has been remedied to some extent by HUBEL & WIESEL (1959, 1962, 1963, 1965a & b) who have studied the receptive fields of single cortical neurones. They have discovered what a cortical cell 'sees' and also how various cortical cells receive their input from other cells to produce a more complex receptive field. Their results have gone some way towards showing how visual patterns are analyzed.

They have studied the light adapted, Nembutal anaesthetized cat and have shown that some cortical neurones in Visual I have receptive fields which show the centre/surround phenomenon as found in the retinal ganglion and LGN cells. On the other hand the shape of the central area is not circular but cigar shaped and the excitatory and inhibitory regions may be asymmetrically arranged. A slit of light covering only the central area will stimulate the cell maximally, a slit perpendicular to this, or diffuse illumination of the whole field will have no effect since both the excitatory and inhibitory areas are being stimulated. Most cells were responsive to movement, if not to a stationary light. The response was sometimes asymmetrical.

These cells were referred to as 'Simple' but there were also complex ones found predominantly in Visual II but also in Visual I.

As with simple cells, slits, edges and bars were effective stimuli and the orientation of the stimulus was critical. The complex cells differed from the simple cells in not being so dependent upon the exact position of the stimulus in the field providing the orientation was correct.

It was suggested (Fig. 10) that the simple cells were composed of a number of similar LGN cell fields, end-to-end, feeding one cortical cell. The complex cells received their input from a number of simple cells, (Fig. 11).



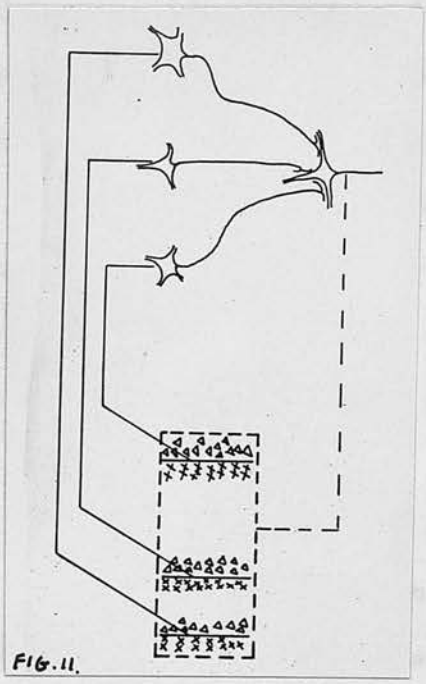


Figure 10
Reproduced from HUBEL & WIESEL (1959) showing how the receptive field of a simple cortical cell might be derived from a number of geniculate cell inputs. + excitatory regions Δ inhibitory regions.

Figure 11
The possible formation of the receptive field of a complex cell from simple ones. (HUBEL & WIESEL, 1962).

The orientation of the cell varied from cell to cell. However, cells with the same orientation were arranged in columns perpendicular to the cortex. Within a column, the field of each cell covered a simi but not identical region of the visual field. Both simple and complex fields were found in the same column but there were fewer

complex than simple cells in layer IV of the cortex. Four-fifths of the cells could be activated by either eye but usually one eye dominated.

These cells had receptive fields through each eye which had the same organisation and axis orientation and were concerned with the same point in visual space (although the accuracy of this last point is limited by the accuracy to which both eyes can be centred). Summation was seen when corresponding parts of the two retinae were stimulated in an identical fashion. Ocular dominance varied from cell to cell. Cells within a column do not necessarily have the same ocular dominance but on the other hand the cells were not scattered entirely at random and neighbouring cells often had similar dominance.

The surface mosaic formed by the intersection of the columnar walls with the cortical surface was highly irregular, although in some parts of the cortex columns were arranged in a regular order. An anatomical basis for these columns is obvious in either Nissl or nerve fibre stained material.

Lower and higher order hypercomplex cells have also been described. A lower order hypercomplex cell also responds to a slit, edge or bar but the stimulus has to be limited in one or both of its ends. That is, it behaved as though it received inputs from two complex cells (or sets of cells), one excitatory to the cell with a receptive field occupying the activating portion, and one inhibitory to the cell, having its field in the antagonistic portion of the receptive field of the hypercomplex cell (Fig. 12).

(see over)

Only a few higher order complex cells were found. Like the lower order cells they required the line stimulus to be limited at one or both ends. Higher order cells differed in responding to

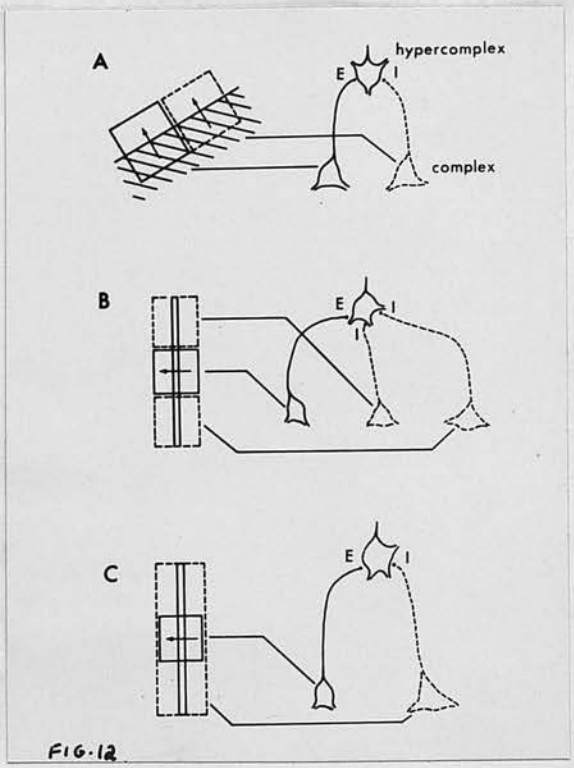


Figure 12.
A. A hypercomplex cell which responds to an edge stopped at one end. It is suggested that the hypercomplex cell receives a projection from two complex cells, one excitatory (E) and the other inhibitory (I).
B. A hypercomplex cell responding to a vertical slit stopped at each end.
C. An alternative scheme to explain the receptive field in B.
(Reproduced from HUBEL & WIESEL 1965).

the line in either of two orientations 90^{o} apart, and the point where the terminus of the line or edge fell within the receptive field was not necessarily critical. These hypercomplex cells behaved as though they received inputs from a large number of lower order hypercomplex cells.

Table I gives the approximate percentages for the distribution of cells within the visual areas.

TABLE 1 Percentage Distribution of Cell Types in the Visual Areas.

	Visual II	Visual III
Simple cells	4	0
Complex cells	90	42
Hyper complex lower order	6	48
Hyper complex higher order	0	10

Since hypercomplex cells had not been recognized when Visual II was investigated the figure of 6% was estimated by Hubel and Wiesel. Figures are not available for Visual I.

Visual II and Visual III were both organized in columns extending from the surface to the white matter, within which there were both complex and hypercomplex cells, all with the same receptive-field orientation, but differing in the precise position and arrangement of receptive fields. In addition, in Visual III there were columns with two receptive field orientations at 90° to each other, containing higher order complex cells which responded to both orientations. In Visual II some groups of columns were arranged in an orderly fashion as one moved across the cortical surface, but in others neighbouring columns were arranged with random shifts of orientation. The distribution of cells according to relative ocular dominance was similar in all three areas.

From a knowledge of the characteristics of the receptive fields of the various types of cells one can imagine what their function might be.

The simple cells of the Visual I and II are likely to respond to contours, breaking down the picture into a series of short lines. Cells entirely within the homogeneous interior of the figure will

not respond as both excitatory and inhibitory regions will be simultaneously activated. Nevertheless the sum of this activity may give some evidence about the brightness of the centre. The other source of information will be the cells which lie half in and half out of the area. This may be a special function of those cells which have only two areas, one excitatory and the other inhibitory, lying side by side forming an 'edge'.

Complex cells will 'see' the same picture but will not be disturbed by a slight movement of the object due to movement of the object or physiological nystagmus of the eye. Segments of curves if appropriately orientated will activate complex units.

This process of abstracting one quality from an area, as opposed to a point in the visual field is taken a stage further with the hypercomplex cells. These will not respond to straight lines or very slightly curved ones. The line must be broken or angled at the end. Hypercomplex cells will see the corners of a square but not the rest of the edges or the centre. Just as these cells will detect corners so they will detect the curvature of a line.

Excellent though their work is, it would be reassuring to have some independent confirmation of the HUBEL & WIESEL findings. BURNS & PRITCHARD (1964) for instance, in the cerveau isolé cat have found cells in the central visual area with much larger receptive fields than Hubel & Wiesel's. ROBERTSON (1965) has found some cells which were silent in the cerveau isolé preparation but which responded to specifically orientated bars after the administration of 'Pentothal'. The receptive fields of another group of cells were considerably altered by the administration of the anaesthetic. This makes one wonder how much of the specificity of the receptive fields described by Hubel & Wiesel is due to their use of anaesthetic But as Robertson points out, the cerveau isolé preparation may not

be comparable with the normal brain, so neither set of experiments may reflect the normal state of affairs.

Whereas Hubel & Wiesel have concentrated on the analysis of pattern by the visual system, BURNS & PRITCHARD (1964) have examined brightness phenomena. They studied the firing pattern of single neurones in the visual cortex of the cerveau isolé cat. Eye movements were prevented by gallamine but the stimuli were moved cyclically to simulate the natural saccades. The average behaviour of the units was determined by computer. Th eir results suggest that a visual neurone indicates which side of a light-dark boundary it represents in terms of the number and sequence of short intervals between action potentials. This is determined by the direction of the intensity gradient across the exciting edge within their receptive field. In other words these cells provide information about contrast and not absolute intensity.

Since most of the stimuli used were edges these neurones could be either simple or complex cells of the HUBEL & WIESEL (1962) classification.

BEHAVIOURAL EXPERIMENTS

Early experiments were made to provide information about measurable visual parameters and this has raised our knowledge from anecdotal level. The work below gives a numerical measure of how small a quantity of light a cat can see. How acute is its vision and if it can discriminate between colours. These are the physical quantities that one can measure but other more complex questions are unanswered. Stimuli which are difficult for a cat to discriminate are obviously different to us. Does this mean that the cat visual system is inferior to ours for pattern vision? Can a cat abstract a small detail which is relevant for a visual discrimination from a mass of irrelevant detail?

Absolute Sensitivity to Light

GUNTER (1951) measured the absolute threshold in six normal cats in a Y-shaped discrimination box. His criterion was an 80% level of success. He found a mean threshold of 9.92×10^{-8} milli-Lamberts (mL) (S.E. 0.92×10^{-8}mL). Two humans under similar conditions had a mean threshold of 5.47×10^{-7} mL (individual results were not given). Thus a cat's eye is at least six times as sensitive as a human eye. BRIDGEMAN & SMITH (1942) found a mean threshold of 8.2×10^{-7}mL (Range 5.8×10^{-8} - 2.4×10^{-7}mL) for 6 cats and MEAD (1942) gives 1.3×10^{-7}mL.

Acuity

SMITH (1936) trained two cats to discriminate between a vertical and a horizontal grid. By reducing the size of the bars until the cats were unable to discriminate he estimated their acuity to be 1.1' for one cat and 0.45' for the other.

Colour Vision

MEYER, MILES & RATOOSCH (1954) were unable to find evidence for this in cats trained for a food reward, providing brightness and colour were equal. However, MELLO & PETERSON (1964) in a more severe experimental situation have demonstrated colour vision. This required long training, starving and an operant procedure.

Various workers have interfered with the brain in an attempt to learn more about its function. They have removed areas and then compared the animals behaviour pre- and post-operatively. This approach is fraught with logical difficulties where the mechanism interfered with is one of many inter-related systems. Under these circumstances the change in the output will not in general, be simply the loss of the contribution normally made by the extirpated area, and the system may now show quite different properties. (GREGORY, 1961). He suggests that the technique is best suited to suggesting or testing possible theoretical models of brain function.

Nevertheless a number of ablation experiments have been made. The results are of some interest, although it is difficult to know what to infer from them beyond saying, this is what happens to an aspect of the animal's behaviour if a particular part of the brain is removed.

SMITH (1937) excised the visual cortex (area 17 and the whole of the lateral and postero-lateral gyri) bilaterally in six cats. Throughout five months observation it was found that they were unable to avoid obstacles, locate food, climb stairs or jump down neatly. They did not blink to a hand thrust in front of the face unless the illumination was very great. The visual placing reaction was lost. Optokinetic nystagmus remained.

DOTY (1953, 1961) has reported that if the striate cortex is removed in new-born kittens that they recover, behave normally and can learn pattern discriminations. This surprising discovery has not been presented in full nor has it been confirmed by other workers.

Absolute threshold

BRIDGMAN & SMITH (1942) found an increase in the absolute threshold to light after removing the 'visual areas'. In one in which a small portion of the gyrus splenialis (i.e. peripheral visual field area 17) remained the threshold had increased five times. In the other, the gyri bordering the lateral and splenial gyri were also removed resulting in a five hundred fold increase.

Differential Brightness Discrimination

Surgical removal of the striate cortex and the ~~part of the~~ lateral and postero-lateral gyrus abolished the ability to distinguish between the two lights of different intensity, when presented in an illuminated surround. There was no deficit if the background illumination was dim (SMITH, 1937). This is also the case in the monkey, where it has also been shown that these discriminations depend on total luminous flux, KLUVER (1942).

Pattern Discrimination is to all intents and purposes lost after removal of the striate cortex and lateral and postero-lateral gyri, (SMITH, 1938).

All the above results imply that the middle suprasylvian gyrus inspite of its visual input (p.44) is incapable of taking over these functions in the absence of the more medial visual areas.

SPERRY, MINER & MYERS (1955) have interfered with the visual cortex in a more subtle fashion by making numerous subpial knife

cuts into the grey matter or by implanting metal wire to interfere with the intragriseal spread of nerve impuses or electrical activity. In some animals the superior colliculi were also removed. They conclude that intragriseal transcortical conduction is not important in view of the high level of post-operative performance.

SPERRY, MYERS & SCHRIER (1960) report an impairment in the 'perceptual capacity of the isolated visual cortex in the cat '. Their method was to lateralize the visual input by ~~either~~ cutting the optic chiasm and masking one eye. All neocortex on one side apart from most of the striate cortex, lateral gyrus, middle suprasylvian gyrus and part of the middle ectosylvian gyrus were removed. There is some doubt in their account of the amount of damage done to the optic radiation as shown by retrograde degeneration in the LGN. The corpus callosum was cut. The cats showed considerable deficits not only in learning visual discriminations but also in their ability to move about the room, jump and find food. However, one can not say on this evidence whether the deficit is in visual perception, attention or the execution of the task. When the cortex was removed in a number of stages the removal of the frontal portion produced a considerable deficit suggesting that this might be an important factor in the syndrome. In monkeys bilateral removal of the prefrontal areas is known to impair their performance in delayed response problems (see WARREN & AKERT, 1964) and BUTLER (~~in press~~ personal communication) has found impairment with a simultaneous visual discrimination problem.

Middle suprasylvian gyrus

HARA (1962) has shown that removal of this region bilaterally has no significant effect on the abilfty of cats to make brightness

discriminations. This contrasts with BADEN et al. (1965) whose removals included the posterior suprasylvian gyrus. There was a moderate but transient impairment in the ability to discrimin ate between figures of different area. They found a marked impairment in the ability to discriminate between a square and a rectangle. While the size of the square was held constant, the size and shape of the rectangle was varied. (This confirms WARREN & SINHA, 1957). There was no impairment in the discrimination between various visual patterns (HARA & WARREN, 1961).

Retention of these problems trans-operatively is not affected but there is significant impairment in post-operative learning of an 'umweg' problem (HARA, 1962; WARREN, WARREN & AKERT, 1961).

Deficits in other modalities have not been looked for.

EXPERIMENTAL INVESTIGATIONS - ANATOMICAL

Two studies were undertaken. One was intended to define the projection of the LGN to the neocortex by means of small stereotaxic lesions and the Nauta stain. The other study dealt with the cortical connections of the visual areas.

No attempt has been made to relate the degeneration to the cytoarchitectonic areas seen on the sections as these boundaries are not distinct (see p.36). Instead, the degeneration has been related to the maps of the electrophysiologists (HUBEL & WIESEL, 1965a; WHITTERIDGE 1966) which have functional significance.

METHODS

Operative technique

The animals were anaesthetized with sodium pentobarbitone ('Nembutal')* in a dose of 0.5 ml/Kg. body weight given intraperitonealy. Additional doses of 0.2 ml. were given if necessary to maintain adequate anaesthesia. The usual aseptic precautions were taken. Those animals which had stereotaxic lesions were given 150,000 units procaine penicillin i.m. at operation.

a) Cat stereotaxic lesions. The needle was inserted through the postero-lateral occipital cortex towards a known thalamic co-ordinate, on a track that was nearly horizontal. This was to avoid the corpus callosum. Before the cat was inserted into the holder, the position of the lesion was determined by clamping the needle drive assembly in approximately the correct line of travel, with the needle fully protruded. The clamp was then adjusted until the needle tip coincided with the tip of a reference electrode lowered in a vertical plane to the appropriate co-ordinate. The reference needle was then removed and the other needle was screwed back so that the cat could

*Abbott Laboratories

be fitted into the holder.

Through a skin incision a small burr-hole was made and the dura opened at the point of entry of the needle. The needle was screwed fully into the brain and a current of usually 0.5 mA was passed for 1/2 minute. The needle was removed and the wound was closed in two layers.

b) Monkey. The procedure was similar except the head-holder was modified to allow the neck to be flexed so that Reid's baseline was vertical. The electrode was then lowered vertically through a burr-hole in the occipital bone, to pass through the cerebellum, entorhinal cortex and hippocampus and so reach the LGN from the ventral aspect.

c) Cortical lesions. The skin was incised and muscle reflected if necessary. The position of the small burr-hole was measured from bregma and the sagittal suture. The hole was carefully made with a dental drill and except in certain cases did not exceed 3 mm. diameter. Looking through an operating microscope, the dura was opened and a small part of the pia was torn away using two pairs of watchmakers forceps. The wound was closed in two layers.

A larger hole was necessary for the lesions made between the hemispheres. This was drilled carefully over the sagittal sinus and for a couple of millimetres over the lateral gyrus on the side on which the lesion was to be made. The dura was cut alongside the sagittal sinus. The brain shrinkage caused by the slow intravenous injection of 20 ml. 30% 'Ureaphil'* given immediately after the anaesthetic was invaluable in allowing a good exposure of the ~~supra~~splenial gyrus without packing or retraction.

Histological technique

After survival periods of 1 - 3 weeks, the animals were anaesthetized with sodium pentobarbitone and perfused with 10% formol

*Abbott Laboratories

saline. In most animals the formol-saline was preceded by 300 ml. 0.9% saline, this was discontinued when it was realised that it was not necessary. The formalin was neutralised with lithium carbonate using bromthymol blue as indicator. This also was not found necessary. Un-neutralised formalin produced just as good results, confirming the observation by NAUTA (1957). The brains were removed and photographed, or drawn to scale. Two days to two weeks later the brains were removed from their pots of 10% formol-saline and cut into convenient blocks. For the LGN experiments, the posterior block extended for 8 mm. from the occipital pole, the middle block measured 15 mm. and the anterior block was the remainder. The latter was not usually stained. For the cortical experiments the block size varied, depending on what was most convenient. The right hemispheres and thalamus were nicked. Coronal sections were cut on a Leitz carbon dioxide freezing microtome, 30μ thick. Three sections out of every twenty cut were saved; through the LGN or the lesion three sections were saved out of every ten cut. Every twentieth section cut was stained with the Nauta method (i.e. about every 0.6 mm.). Every tenth section through the lesion or LGN was stained with a Nissl stain, usually cresylviolet, but sometimes thionin or chrome alum gallocyanin. See appendix for details. Spare sections were available if a repeat staining was necessary.

A multi-compartmented sieve was designed to process the sections for the Nauta method. This had the advantage that the loose frozen sections were kept in strict serial order and were not damaged by tedious, individual handling. Details of the sieve and the staining procedure are given in the appendix (p.142).

RESULTS

Projections from the LGN in the cat and monkey

The following experiments show that the LGN projects to three

separate ipsilateral areas of the cat neo-cortex. These comprise Visual I and Visual II of TALBOT (1942), BILGE, SENEVIRATNE & WHITTERIDGE (1963) and HUBEL & WIESEL (1965), and thirdly to the lateral edge of the middle suprasylvian gyrus. Various control lesions have been made to determine whether the projections to Visual II and the suprasylvian gyrus arise in the LGN or in a neighbouring structure. The monkey LGN projects only to the ipsilateral striate cortex.

The cat brains were cut into coronal blocks by cuts 8mm and 23mm in front of the occipital pole. The posterior block contained the point of entry of the needle electrode, the middle block had most of the cortical fibre degeneration, and the anterior block was substantially free from degeneration in the two brains in which it was studied. Since in the cat the needle electrode entered the neocortex and passed through the white matter before reaching the diencephalon, it was important to know what cortical fibre degeneration would be produced by the needle track alone if no thalamic lesion was made. Fig. 13 shows a typical track through the brain. It should be noted that the first thalamic structure encountered by the needle is the posterior end of the LGN.

Control lesions

In two cats (C1, C2) the needle was inserted just short of the LGN and no current passed, while in three other cats (C3 - C5) the needle was misplaced in the third ventricle when the current was passed. In the posterior block (as defined above) degenerated fibres were found spreading from the needle track to the inferior half or three-quarters of the postero-lateral gyrus on both its lateral and medial surfaces, and to the posterior suprasylvian gyrus. In C1 there was a moderate amount of fibre degeneration in the crown only of the middle suprasylvian gyrus extending forwards into the middle block. The lateral gyrus was however, clear of degeneration in the

middle block, and in the other four brains (C2 - C5) the whole of the middle block was clear of degeneration apart from an occasional degenerated fibre on the lateral gyrus. No reason has been found for the degeneration in C1 being more extensive than in C2 - C5.

In two other cats (C12, C18) the needle track ended in a thalamic lesion (Details given later under C12, C18),, but there was no fibre degeneration in the cortex of the lateral or middle suprasylvian gyri more than 12 mm. in front of the occipital pole. In addition to the inadvertant damage done by the needle track in the white matter, the cortex at the point of entry was sometimes damaged in the superficial layers. In two further cats (C8, C9) the relevant superficial layers of cortex were damaged deliberately without inserting a needle. One of these small lesions was confined to the postero-lateral gyrus, and the other to the posterior suprasylvian gyrus, but in both brains the only degenerated fibres found were localized to the immediate vicinity of the lesion.

Most of the information to be presented was derived from the study of the cortex in the middle block, 8 - 23 mm anterior to the occipital pole. The results in the control brains may be summarized (Fig. 14) by saying that the lateral gyrus from 12mm ahead of the occipital pole was free from degeneration in all 9 of the control lesions, while in all 7 of the control lesions without thalamic involvement it was free throughout the middle block. The middle suprasylvian gyrus was similarly free from degeneration except in one brain in which the crown but not the walls of the gyrus contained degeneration. The posterior block however contained degeneration caused by the needle track which must be allowed for in drawing conclusions from the results of thalamic lesions.

LGN lesions (excluding the medial interlaminar nucleus)

Three cats (C10 - C12) had thalamic damage entirely confined to the LGN, while in others there was additional involvement of medially

placed structures. The three lesions to be described were small enough to produce an interesting topographical distribution of degeneration in the visual cortex. To understand this it is necessary to remember that the vertical meridian of the visual field is represented on the medial edge of the LGN (BISHOP, KOZAK, LEVICK & VAKKUR, 1962; SENEVIRATNE & WHITTERIDGE, 1962). BILGE et al. (1963) and HUBEL & WIESEL (1965) have shown that the vertical meridian on the cortex is represented by the boundary between Visual I and Visual II and that this boundary coincides between that of the area striata (17) and the area occipitalis (18) as defined by OTSUKA & HASSLER (1962). Furthermore, the anterior part of the LGN projects to the anterior visual cortex, and the posterior part of the LGN posteriorly (MINKOWSKI, 1913).

In the first cat (C10), the lesion extends from postero-lateral to antero-medial LGN (Fig. 15), and thus affects the representation of the peripheral field at the posterior end of the LGN, but the central field at the anterior of the LGN. In the cortex, two areas of degeneration are found, one on the medial wall of the hemisphere in area 17 or Visual I, and the other on the top of the lateral gyrus in area 18 or Visual II. In the posterior part of the middle block, a degeneration-free zone intervenes between these two areas of degeneration which are widely separated, as are the representations of the peripheral field in Visual I and Visual II at this level which is anterior to the representation of the area centralis. Anteriorly, the two areas of degeneration come together at the representation of the vertical meridian on the lateral side of Visual I and the adjoining medial side of Visual II (see Fig. 15). It will be argued that this topographical correspondence between the distribution of the cortical fibre degeneration and the position of the lesion in the LGN is a strong reason for thinking that the cells of the LGN project to the two areas Visual I and Visual II, and that the result is not due to damage to fibres of other origin which happen to be passing near the

lesion in the LGN.

In C10 no degeneration was found on the medial lip of the lateral sulcus corresponding to area praeoccipitalis (19), the Visual III of the electrophysiologists. However, a sparse but definite projection was found to the lateral wall of the middle suprasylvian gyrus, a region that was clear of degeneration in all the control brains. In the three areas of degeneration described above, the broken fibres were of similar medium calibre, and were densest in layer IV. There was little in layer III and even less more superficially.

In the second cat, C11, the lesion was on the lateral edge of the LGN, and passed forwards to the anterior extremity of the nucleus with some possibility of damage to the reticular nucleus just beyond, and to one small bundle of fibres from the nucleus lateralis posterior. This then was a lesion in the representation of the far peripheral visual field. In the striate cortex the fibre degeneration was ventro-medial near the splenial sulcus (Fig. 16). A second patch of degeneration appeared on the lateral half of the lateral gyrus. Since Visual II and Visual III abut in this region, it is difficult to say whether the degeneration observed contained a separate projection to Visual III in this brain. Definite fibre degeneration was again seen in the lateral wall of the middle suprasylvian gyrus, and scanty degeneration also occurred in the adjoining medial wall of the ectosylvian gyrus. The last finding and the possibility of damage to the reticular nucleus and nucleus lateralis posterior in this brain are the only respects in which these results differ from the pattern set by C10.

In the third cat, C12, the lesion damaged the postero-dorsal part of the LGN and produced fibre degeneration in the postero-lateral and posterior suprasylvian gyri that was denser than in the

control brains. Some degeneration also occurred in the lateral wall of the middle suprasylvian gyrus, and in the medial wall of the ectosylvian gyrus. This result is compatible with the findings in C10 - C11, but it is of limited value because the posterior placement of the lesion caused the cortical degeneration to occur in the posterior block.

Two of the cats are of some help in demarcating the visual areas. In C10 the lesion extends to the antero-medial tip of the LGN. Consequently, the most anterior degeneration seen on the cortex (see Fig. 15) corresponds to the anterior limit of the visual area. The lesion in the lateral tip of the LGN in C11 (Fig. 16) produces degeneration in the upper wall of the splenial sulcus, extending almost to the bottom of the sulcus. This is likely to represent the extreme periphery of the visual field, as the peripheral visual field is represented laterally on the LGN (BISHOP et al.1962; SENEVIRATNE & WHITTERIDGE, 1963).

Lesions of structures medial to the LGN

The surprising finding that the LGN appears to project to Visual II as well as to Visual I, makes it desirable to determine whether these degenerated projections could have arisen from structures bordering the LGN. Examination of Nissl-stained sections of the thalamus did not reveal any retrograde reaction in the nuclei medial to the LGN in any of these experiments. To be certain, lesions were placed in these more medial structures to see whether the resultant pattern of degeneration would fit that seen after lesions of the LGN.

One cat (C13) had a lesion that was centrally placed at the posterior end of the LGN, but that moved medially further forwards to involve the medial interlaminar nucleus of the LGN. In the cortex degnerated fibres were distributed as an almost continuous band in the dorso-medial part of the lateral gyrus, as would be expected of a lesion close to the representation of the vertical meridian. However, degenerated fibres were also present in the lateral wall of the lateral gyrus, in a region that may be either area 18 or 19. Some degeneration was again present in the lateral wall of the suprasylvian gyrus in the middle part of its antero-posterior extent. These results are similar to those obtained in C10 except for the added degeneration in the lateral wall of the lateral gyrus, and the added involvement of the medial interlaminar nucleus in the lesion.

In cat C14 (Fig. 17a) a lesion was made within the posterior half of the pulvinar nucleus with involvement of the medial interlaminar nucleus of the LGN. No retrograde cell reaction was seen in the LGN in Nissl-stained preparations. No fibre degeneration occurred in the dorsal or medial parts of the lateral gyrus (area 17), but degeneration in the lateral half of the lateral gyrus extended down into the depths of the lateral sulcus. In the suprasylvian gyrus, degen-

eration was moderate on the crown, but became dense in the lateral wall. There was also moderate degeneration in the medial wall of the ectosylvian gyrus, and a small patch at the bottom of the splenial sulcus. The last named region had been free of degeneration in the brains described above, with the possible exception of C13 : no such degeneration was seen in this brain, but the medial interlaminar nucleus was damaged anteriorly, and there was a gap in the series of sections of this brain at an anterior level.

In C15, a small lesion damaged the medial interlaminar nucleus of the LGN and part of the N. posterior and N. lateralis posterior. Cortical fibre degeneration was found in the lateral two thirds of the lateral gyrus extending down the lateral wall of the gyrus, in the lateral half of the middle suprasylvian gyrus again extending down the wall, and in a small patch at the bottom of the splenial sulcus. Scant scattered degeneration occurred elsewhere in the medial wall of the hemisphere (area 17) and at the tip of the medial wall of the ectosylvian gyrus. The degeneration in area 17 was probably due to the passage of the needle track through the central part of the LGN. Degeneration found on the posterior ectosylvian gyrus is probably due to track damage in the white matter.

In C16 the lesion penetrated the medial interlaminar nucleus ~~(Fig. 17e)~~ and extended as for forwards as the N. ventralis anterior, causing also some damage in the pulvinar nucleus. The pattern of degeneration was similar to that in C15, except that degeneration in the medial wall of the hemisphere was confined to a small patch at the bottom of the splenial sulcus. These four brains (C13 - C16) had in common damage to the medial interlaminar nucleus of the LGN and all possessed a degenerated projection to the lateral part of the lateral gyrus, which was not degenerated in the three brains (C10 - C12) with pure lesions of the LGN. Three brains (C14 - C16) also showed degeneration in a small region at the bottom of the splenial

sulcus. This latter region and the lateral part of the lateral gyrus have in common that they contain part of area 18 or 19 or both. It is however, difficult to say whether the regions receiving the degenerated projection should be designated as area 18 or 19 (see Discussion).

In another cat, C17, the lesion damaged mainly the pulvinar nucleus, with some involvement of the N. lateralis posterior and N. lateralis dorsalis, but did not touch the LGN or its medial interlaminar nucleus (Fig. 17c). The cortical degeneration was confined to the crown and lateral wall of the suprasylvian gyrus in the middle part of its antero-lateral extent. The lateral gyrus was entirely free from degeneration.

A lesion that extended most of the length of the N. lateralis posterior was made in another cat (C18) without involvement of other nuclei. No retrograde reaction was seen in Nissl preparations in the LGN or pulvinar nucleus. Fibre degeneration was found only on the posterior ectosylvian and posterior suprasylvian gyri, and was probably due to track damage. WALLER & BARRIS (1937) suggested that N. lateralis posterior projects to the anterior end of the suprasylvian gyrus. Unfortunately, the frontal block was not stained. The results in C18 do however exclude a projection to the lateral gyrus from this source.

Combined lesions of the LGN and more medial structures

In the course of making the lesions described above, seven other brains (C19 - C25) were studied in which a lesion in the LGN had spread to involve more medial structures including the medial interlaminar nucleus and the pulvinar nucleus. In one brain (C19) the lesion was just beneath the LGN, damaging the latter medially and involving the medial interlaminar nucleus, and part of the posterior

nucleus, N. lateralis posterior and perhaps part of the pulvinar nucleus. Dense degeneration was distributed laterally across the lateral gyrus from its medial edge, but did not descend far into the wall of the gyrus. There was also some slight degeneration on the medial wall of the hemisphere especially at the bottom of the splenial sulcus, and in the middle suprasylvian gyrus. In the other six cats the lesion involved the LGN dorso-medially, as well as the interlaminar nucleus and other medial structures. Two of these lesions were so far posterior that the degeneration in the cortex was confined to the posterior block where it was mixed with degeneration due to the needle track through the white matter. In the other four brains (C20 - C23) there was degeneration in the middle suprasylvian gyrus and in the lateral part of the lateral gyrus, and at the bottom of the splenial sulcus. Besides this constant finding, there was degeneration in the crown of parts of the lateral gyrus corresponding to the variable antero-posterior placement of the lesion in the LGN.

Contralateral degeneration

All the fibre degeneration described above was ipsilateral with the lesion, and no contralateral degeneration was seen in any of the 11 brains with lesions of the LGN (C10,11,12,13,19 - 25), although occasional degenerated fibres were found in the posterior block at the position corresponding to the entrance of the electrode. These are likely to be transcallosal cortico-cortical fibres (EBNER & MYERS, 1965). The lesions confined to more medial structures did not cause contralateral degeneration either. The needle track was fine (Fig. 13b) and avoided the corpus callosum (Fig. 13a). The range of survival times covered the period of 1 - 3 weeks specified by GLICKSTEIN, MILLER & SMITH (1964), who had described a projection from the LGN to the contralateral lateral gyrus through the corpus callosum.

The findings in the cat are summarised in Fig. 18.

PROJECTIONS FROM THE LGN IN THE MONKEY

In view of these interesting findings it seemed worthwhile repeating the experiment in the monkey to see if it had more than one cortical projection from the LGN. Furthermore it was important to find any contralateral projection as this would have serious consequences for 'split brain' experiments (e.g. DOWNER, 1961a).

In the first monkey, there was a thin needle track through the cerebellum, entorhinal cortex, subiculum and fimbria, ending in a lesion 1.5 mm in diameter which affected the postero-dorsal pole of the LGN and the inferior part of the pulvinar nucleus. The occipital lobes were studied in parasagittal section. In the cortex, there was dense fibre degeneration in the antero-ventro-lateral part of the striate area that abuts the lunate sulcus and lies immediately dorsal to the inferior occipital sulcus (i.e. the macular region). This patch of degeneration was entirely confined to the cortex containing the stria of Gennari, and did not extend forwards into the peristriate cortex. The cortical degeneration was ipsilateral to the lesion, all parts of the contralateral striate and peristriate cortex being clear of degeneration.

In the second monkey, there was a similar needle track but the lesion was further forwards in the middle of the antero-posterior extent of the LGN. The lesion was 1 mm in diameter and entirely confined to the lateral limb of the LGN. In the occipital lobes, which were cut frontally, there was dense fibre degeneration in the ventral wall of the calcarine sulcus on the medial side of the hemisphere at the posterior end of the brain (i.e. degeneration was in the representation of peripheral visual field). The degeneration was again confined to the striate area, and none was seen in the peristriate cortex, or in the occipital lobe contralateral to the

lesion.

In both monkeys the degeneration extended to the edge of area 17 so that one would expect the related area 18 to be adjacent. This region was scrutinised without finding a second area of degeneration.

A further observation was that the striate of Gennari was relatively free of degeneration, confirming CLARK & SUNDERLAND (1939) that the stria is not composed of dispersing radiation fibres. The degeneration was found predominantly in the layers immediately above and below the stria.

Figure 14

A composite representation of the distribution of cortical fibre degeneration in the control brains C2 - C5, C12, C18. The dotted lines show where the brains were cut into posterior, middle and anterior blocks. The needle electrode entered the cortex within the region of the black circle.

Figure 15

(a) The distribution of cortical fibre degeneration following an LGN lesion in C10. Note the wide separation of the two medial projections at the posterior end of the middle block and their convergence at the representation of the vertical meridian anteriorly. The suprasylvian degeneration in the wall of the gyrus is shown in the cross-sections.

(b) The site of the lesion at selected levels through the thalamus. (Jasper & Ajmone-Marsan 1955).

Figure 16

The distribution of cortical fibre degeneration in C11 at three levels following a lesion in the lateral margin of the LGN.

Figure 17

A A cresyl-violet preparation to show the lesion in the medial interlaminar nucleus of the LGN in C14 and B the distribution of cortical fibre degeneration caused by this lesion.
C shows the distribution of cortical fibre degeneration following the pulvinar lesion in C17.

The site of the lesions in the thalamus are shown on the adjacent page.

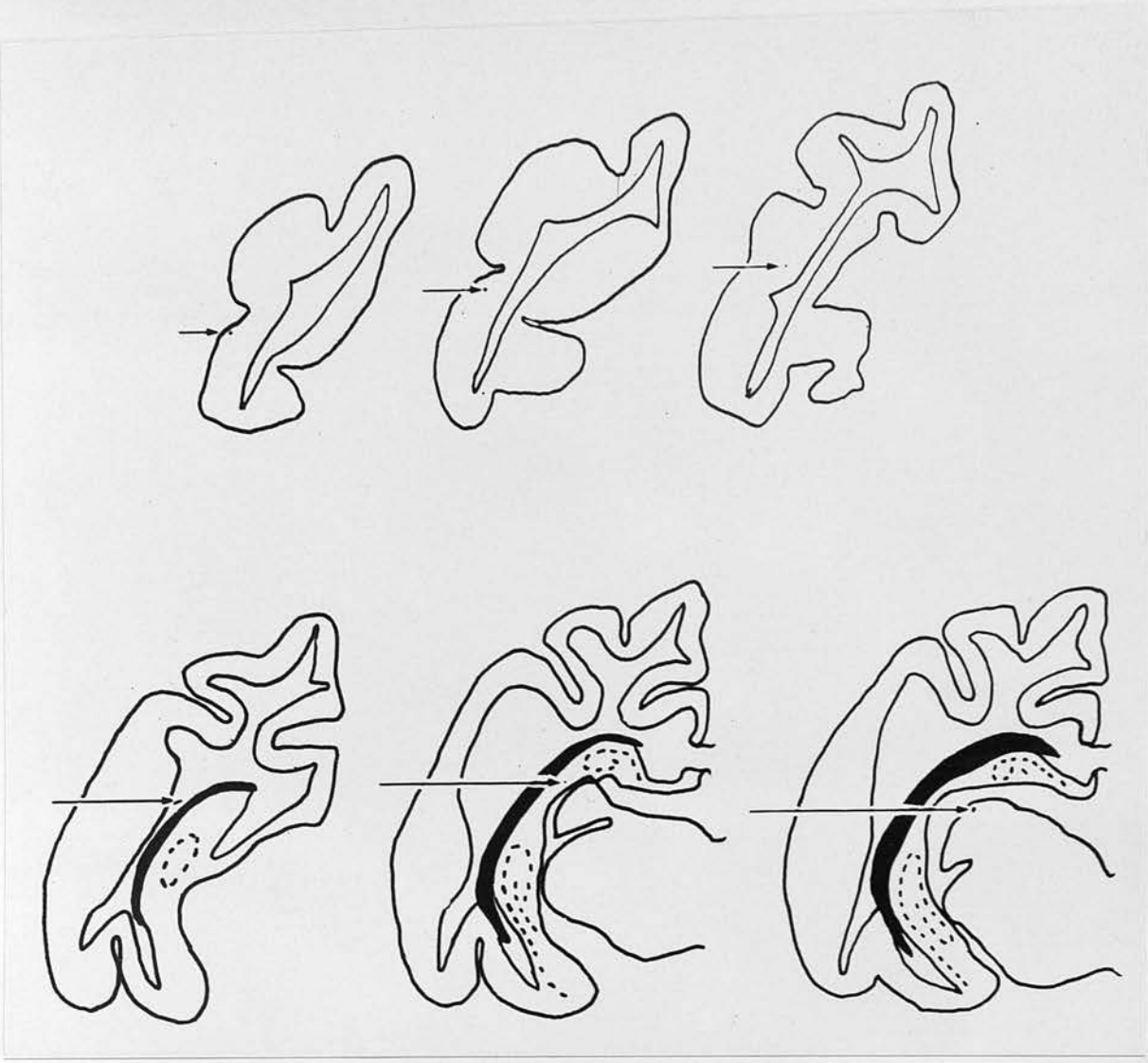


FIG 13 A

B

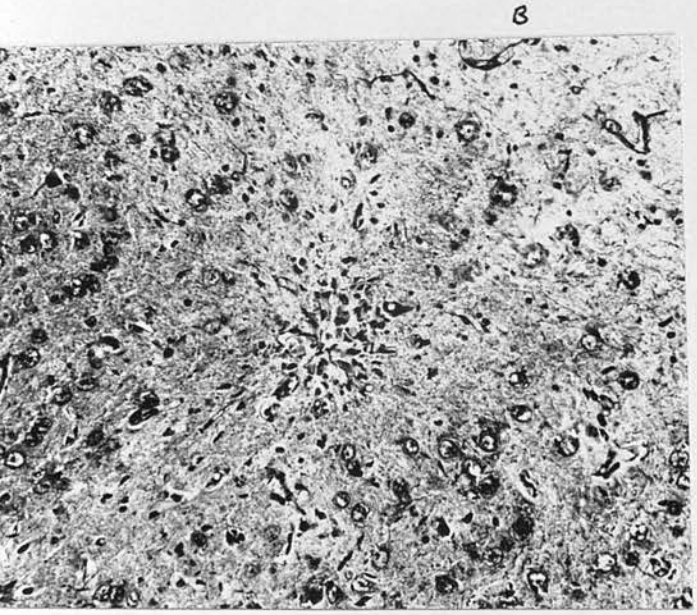


Figure 13

A The position of a needle track aimed at the LGN, showing the structures traversed at six level and B the size of a typical needle track in a Nauta preparation.

100 μ.

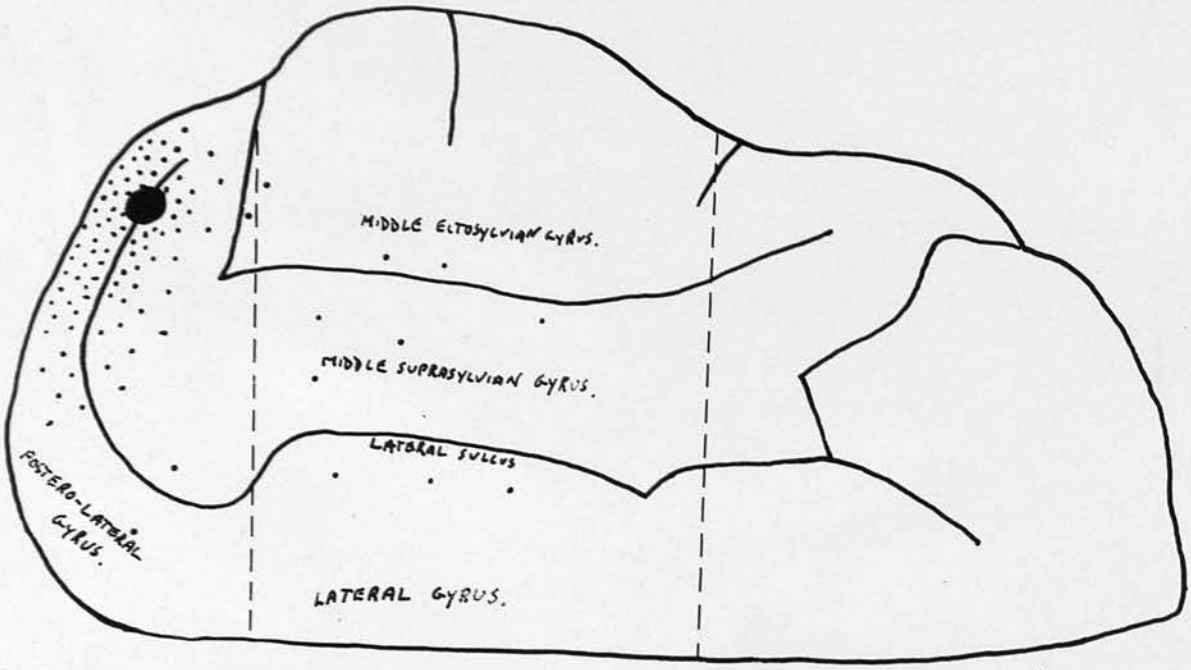


FIG. 14.

C10

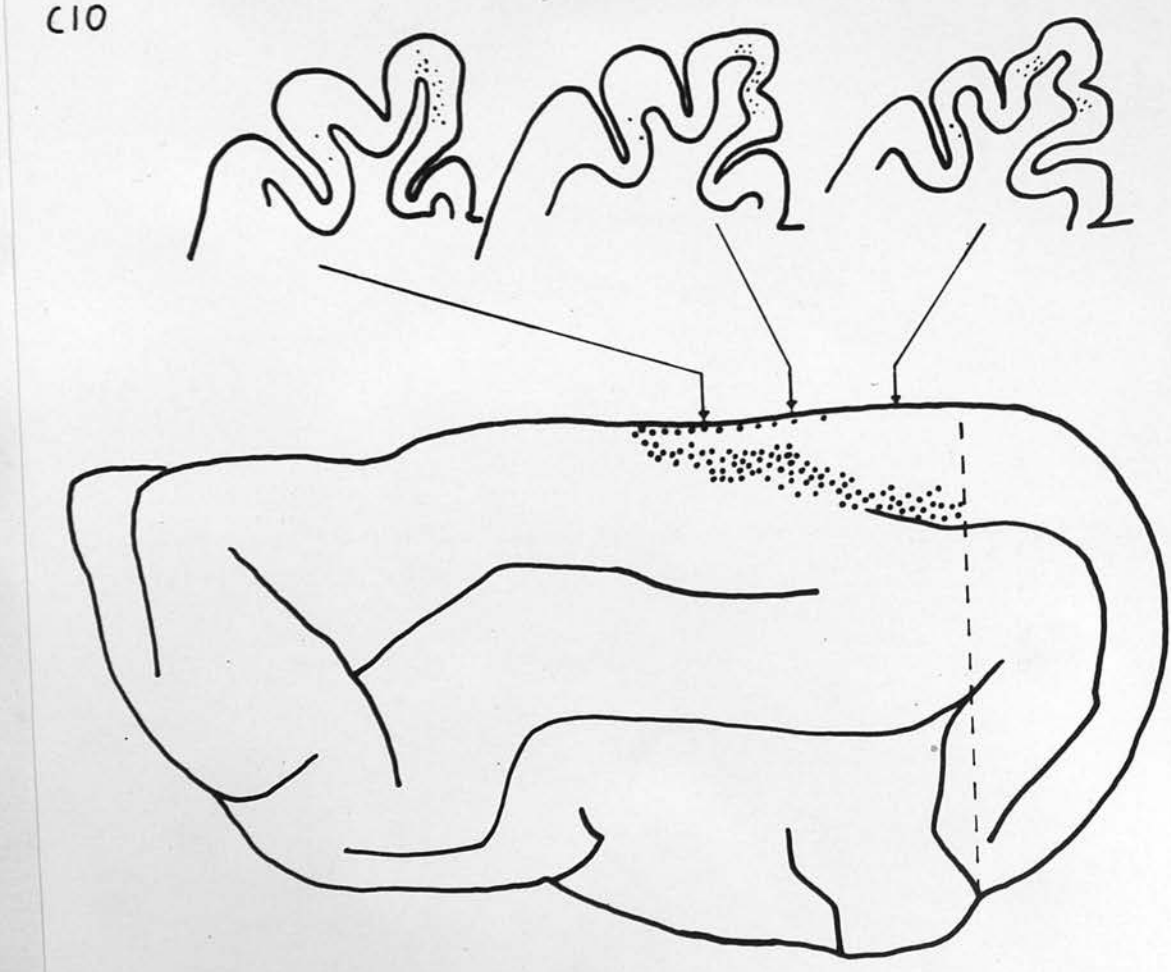


FIG. 15. (a)

C10

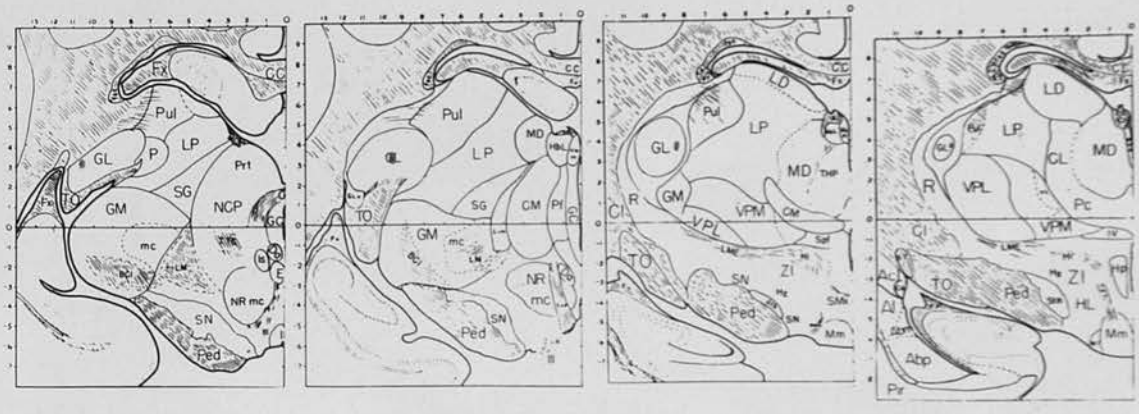

Fx
CC
Pul
GL
P
LP
Pr1
TO
GM
SG
NCP
mc
NR
SN
Ped
MD
CM
Pf
LD
R
VPL
VPM
ZI
Cl
Pc
HL
Mm
Abp
Fr.5-0
Fr.6-5
Fr.8-0
Fr.9-0

FIG. 15(b)

C11

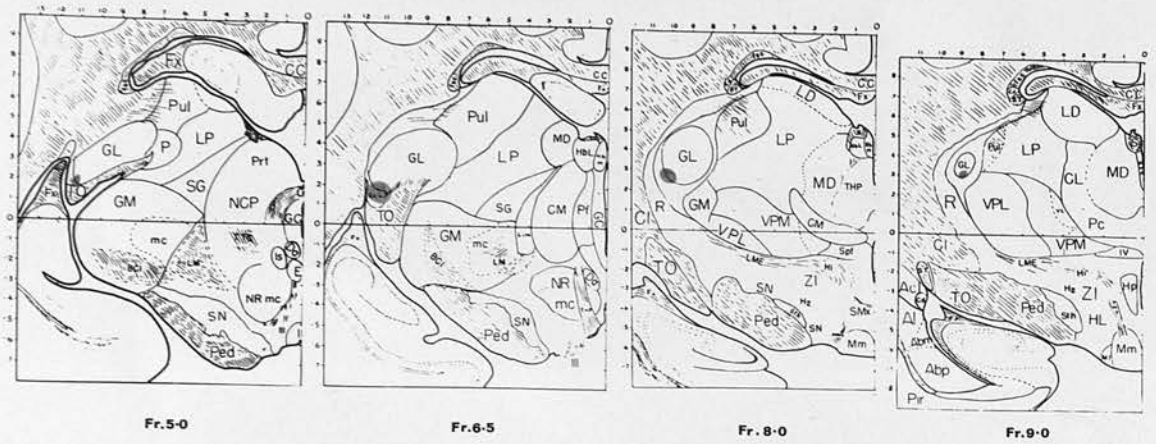


FIG. 16.

C14

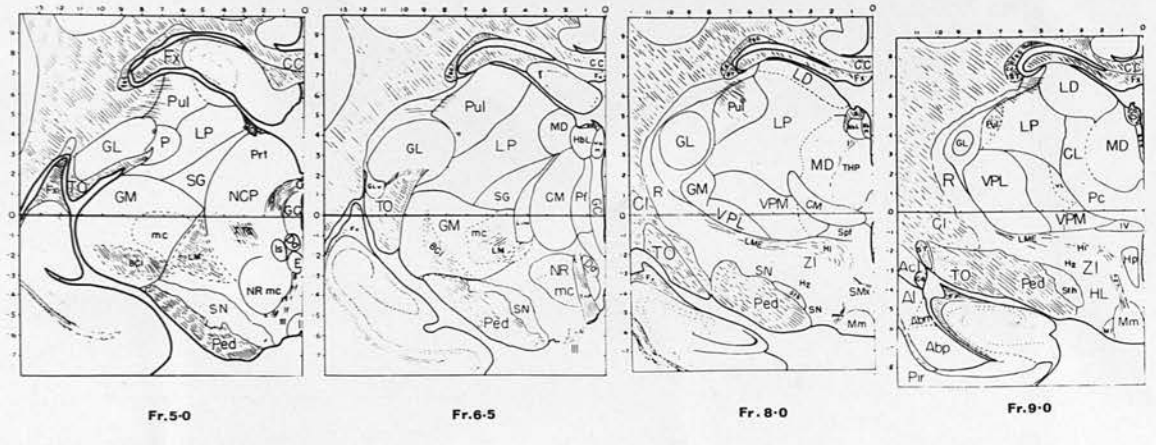


FIG 17a

C17

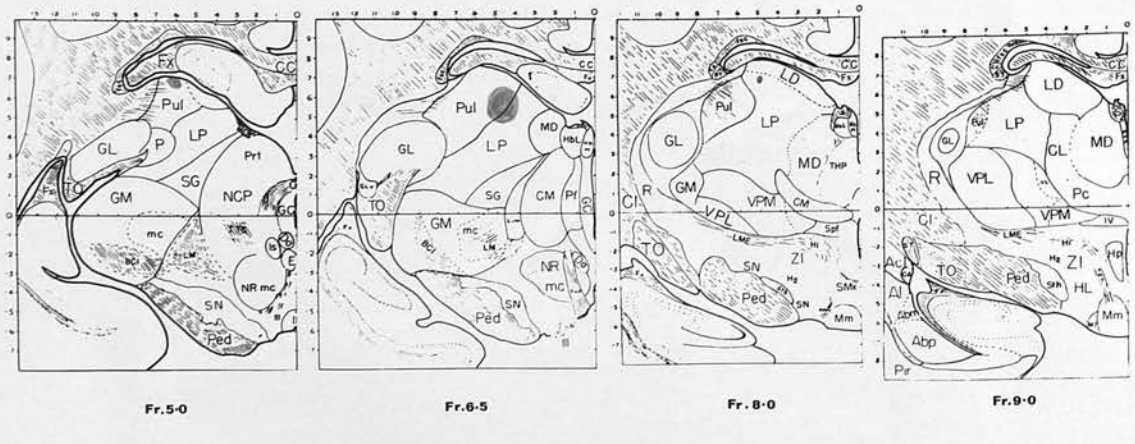


FIG. 17 c.

FIG. 16

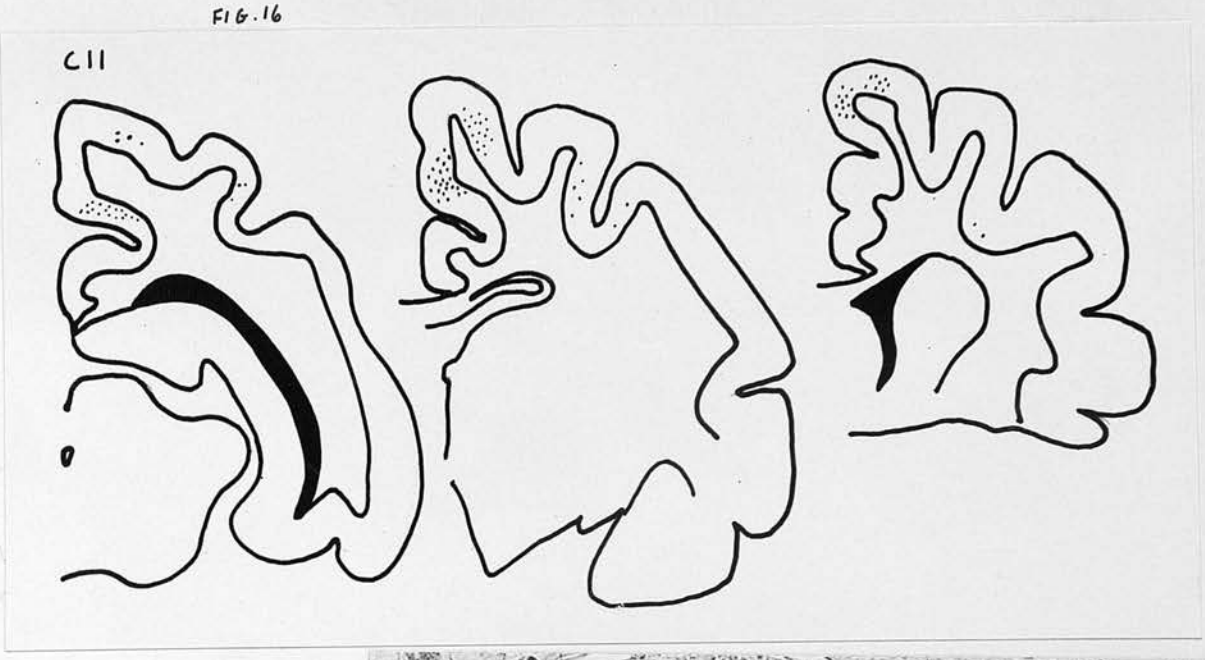
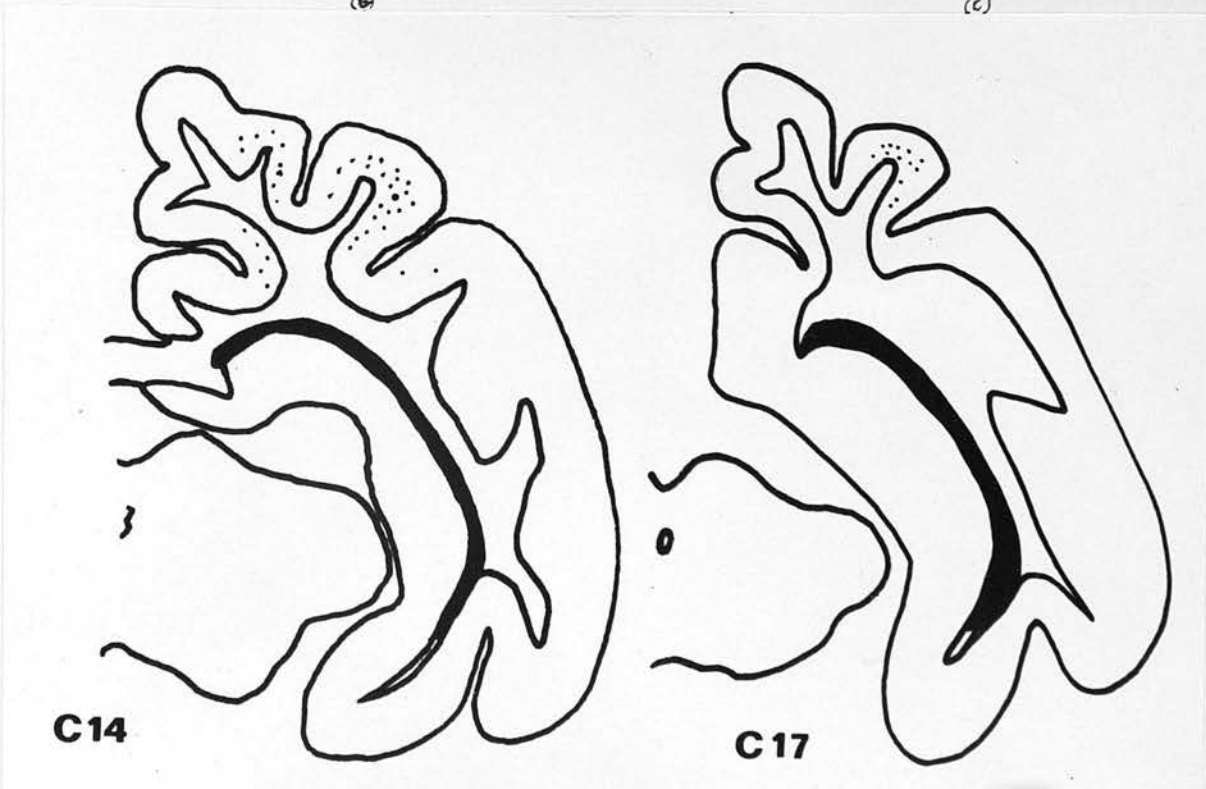


FIG. 17.



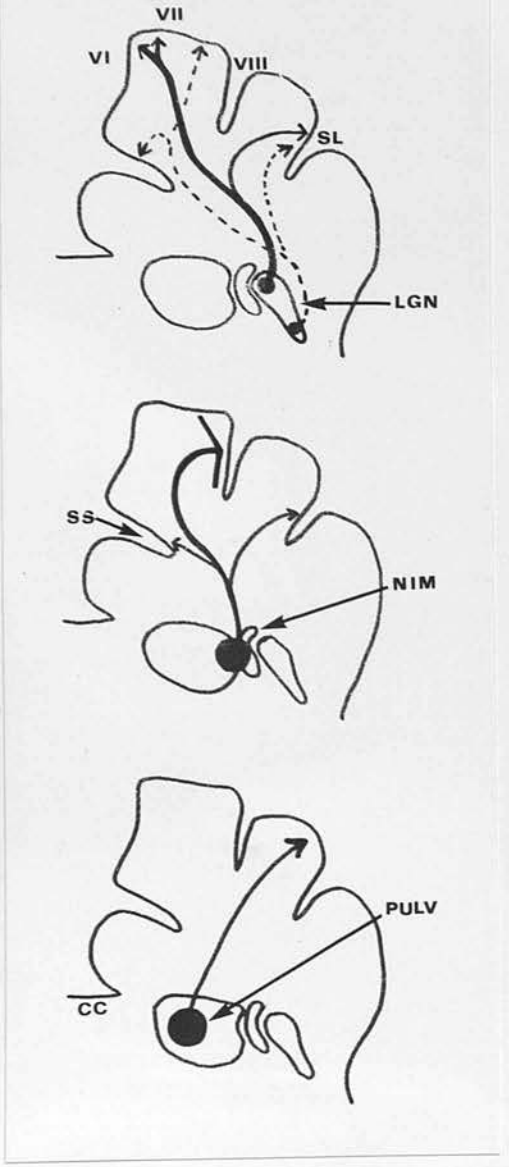


FIG. 18.

Fig. 18. A summary diagram to show the distribution of cortical fibre degeneration following lesions in the antero-medial and postero-lateral parts of the LGN; in the structures immediately medial to the LGN, including the medial interlaminar nucleus (NIM); and in the more medial structures including the pulvinar nucleus (PULV). VI, VII & VIII are the visual fields, SL the lateral part of the middle suprasylvian gyrus, SS the splenial sulcus and CC the corpus callosum.

DISCUSSION

General Comments on the Nauta Method

The Nauta method has its disadvantages and it can be capricious. The degree of suppression is critical, if this is excessive some of the degenerated fibres will be lost. Conversely too little suppression brings out too many normal fibres and if too much silver is used in the ammoniacal silver bath it will be very difficult to detect the degenerated fibres amongst such a dark and dense background. However, optimal suppression was usully possible by the adjustment of the ammoniacal silver bath. If this failed a fresh batch of sections were stained with a longer or shorter time in potassium permanganate.

It is possible that subjective factors might influence the pattern of degeneration reported. There was some check on this, as occasional sections were mounted laterally inverted. It was reassuring to be startled by one of these slides when one found degeneration appropriate to say the side ipsilateral to the lesion when one thought one was looking at a contralateral side.

In spite of these cautionary remarks the average Nauta stain was of good quality and degeneration could be reliably plotted. In the occasional truly excellent Nauta preparation, both positive and negative findings are indisputable.

The Projection of the Lateral Geniculate Nucleus

The results in the cat show that the striate area receives a topographically organised projection from the LGN, for medial lesions in the latter produce fibre degeneration in the lateral edge of area 17, while a lateral lesion (C11) caused fibre degeneration deep in the medial wall of the lateral gyrus (see Fig. 16). This arrangement is consistent with the results of earlier workers who studied

the location of retrograde degeneration in the LGN after making lesions in parts of the lateral gyrus (MINKOWSKI, 1913; WALLER & BARRIS, 1937). The mapping of the visual field by electrophysiological methods in the LGN (SENEVIRATNE & WHITTERIDGE, 1962; BISHOP KOZAK, LEVICK & VAKKUR, 1962) and on the visual cortex (TALBOT & MARSHALL, 1941; BILGE, SENEVIRATNE & WHITTERIDGE, 1963) again implies the same topographical relationship between the LGN and the visual cortex.

In the monkey, the first lesion destroyed the postero-dorsal pole of the LGN, and produced fibre degeneration in the antero-ventro-lateral corner of the striate cortex in the angle between the lunate and inferior occipital sulci. This result is also consistent with previous work, for CLARK & PENMAN (1934) found that a lesion in the macular part of the retina produced transynaptic neuronal atrophy in the postero-dorsal pole of the LGN, and TALBOT & MARSHALL (1941) recorded electrical responses in the antero-ventro-lateral part of the striate cortex to photic stimulation of the macula. Degenerated fibres in the striate cortex after lesions in the LGN were not concentrated in the stria of Gennari, and this is consistent with the preservation of the latter after under-cutting the striate cortex, as found by CLARK & SUNDERLAND (1939). In both monkeys with LGN lesions, fibre degeneration in the striate area extended to the boundary of areas 17 and 18. MYERS (1965) showed that this part of the striate area is connected to the adjacent part of area 18. The abrupt cessation of the degeneration at the boundary of area 17 with area 18 was thus convincing evidence that the LGN in the monkey does not project into the appropriate part of area 18. Fibre degeneration was moreover absent from the rest of areas 18 and 19.

The finding in the cat of a second projection from the LGN that supplies afferents to area 18 was unexpected, although this area was

known to contain a second topographical representation of the visual field (Visual II) as shown by TALBOT (1942) and BILGE, SENEVIRATNE & WHITTERIDGE (1963). Indeed, TALBOT (1942) suggested that Visual II received an independent projection from the LGN, for the responses there were not 'depressed by narcosis or cautery of the lateral gyrus', and were 'independent of convulsants applied at the medial locus'. Moreover, DOTY (1958) was able to record photic responses from the lateral half of the lateral gyrus after the more medial area had been removed. However, removal of the lateral area (containing Visual II) produced only a small area of retrograde degeneration in the medial edge of the LGN. This degeneration was attributed to the lesion extending into the optic radiation running to the striate cortex (DOTY, 1958).

There is a possibility that the projection to Visual II degenerates after the lesions in the LGN because of damage to fibres of extraneous origin which happen to be passing through the LGN. There are, however, two facts that make this explanation improbable: first, the disposition of the degeneration in Visual II is topographically organised in a manner complementary to that in Visual I (see Fig. 16), being thus compatible with the mapping of the visual field in Visual II (WHITTERIDGE, 1966). Fibres arising in more medial structures and passing through the LGN towards the internal capsule would be interrupted equally by medial or lateral lesions in the LGN, and would not therefore be expected to show a topographical distribution related in the correct manner to the position of the lesion in the LGN. Secondly, lesions have been made in the structures surrounding the LGN medially, and the resulting cortical degeneration in the lateral part of the lateral gyrus only slightly overlapped that found in Visual II after LGN lesions, and never extended to the medial boundary of Visual II where central vision is represented and where degeneration was found

after lesions in the medial part of the LGN.

These two arguments do not apply to the cortical degeneration seen in the suprasylvian gyrus after LGN lesions, for this was also present after lesions in more medial structures and did not show any marked topographical distribution. There is however, independent electrophysiological evidence of a direct projection from the LGN to the suprasylvian gyrus (VASTOLA, 1961) which has been corroborated by the evidence of THOMPSON et al. (1963). BUSER et al. (1959) have shown that a pathway via the thalamus medial to the LGN may be important for photically evoked responses on the crown of the middle suprasylvian gyrus. They did not show that this pathway is essential, and in any case they may have been dealing with an area of the suprasylvian gyrus more medial than that implicated in the experiments with LGN lesions. BRUNER (1965) has reduced but not abolished the photically evoked response in the lateral part of the middle suprasylvian gyrus by injecting potassium chloride into the thalamus medial to the LGN. No studies of retrograde degeneration in which only the middle suprasylvian gyrus was removed have been reported. MARSHALL et al. (1943) in one cat removed the lateral gyrus on one side and, after allowing time for retrograde degeneration to occur, were unable to obtain the short latency response from the middle suprasylvian gyrus. It is unfortunate that no histology was reported, as this result may imply that the middle suprasylvian gyrus is supplied by collaterals of fibres to the lateral gyrus, and that these collaterals are insufficient to sustain the parent cell body when the main branch to the lateral gyrus is destroyed.

In addition to these three projections from the LGN discussed above, degenerated fibres were found in the lateral half of the lateral gyrus after lesions of structures medial to the LGN. This cortical degeneration seemed to be mainly in area 19, or Visual III of WHITTERIDGE (1966) and HUBEL & WIESEL (1965), with perhaps some

extension into the lateral part of area 18 or Visual II. When degeneration was present in the lateral part of the lateral gyrus, it also appeared at the bottom of the splenial sulcus. Although this degeneration extended some way along the upper wall of the sulcus, it was still more deeply situated than that caused by the most lateral lesion in the LGN. By analogy with the situation on the dorsal surface it would appear that this is a projection to either area 18 or 19 related to the extreme periphery of the visual field. Cytoarchitectonic studies of this region are conflicting : WINKLER & POTTER (1914) and GUREWITSCH & CHATSCHATURIAN (1928) described an area 18 here, but OTSUKA & HASSLER (1962) distinguish area 18 only on the dorsal surface of the brain, and describe the region in the depth of the splenial sulcus as area 19. However it is difficult to imagine Visual II or III (area 18 or 19) in the splenial sulcus in the topographical scheme proposed by WHITTERIDGE (Personal communication - see Fig. 6).

The alternative hypothesis that the degeneration in the splenial sulcus lies in Visual I can not be supported for topographical reasons either, even allowing that there may be no representation of central vision in the medial interlaminar nucleus (STONE & HANSON, 1966).

A third possibility is that the degeneration in the splenial sulcus is neither in Visual I, II nor III but in some other region, perhaps cingulate cortex.

The exact origin of this projection from structures medial to the LGN is uncertain. The relevant lesions always involved the medial interlaminar nucleus, but also extended into adjacent parts of the pulvinar nucleus. One medially placed lesion (C17) was restricted to the pulvinar, lateralis dorsalis and lateralis posterior nuclei, and cortical fibre degeneration was confined to the middle suprasylvian gyrus (see Fig. 17c). GAREY (1965) has reported retrograde degeneration in the medial interlaminar nucleus following cortical

lesions placed lateral to area 17 of OTSUKA & HASSLER (1962). These results suggest that the medial interlaminar nucleus may be the origin of the projection to the lateral part of the lateral gyrus (Visual III) but the possibility cannot be excluded that the most medial part of the pulvinar nucleus untouched by the lesion in C17 might also project to Visual III. Whether the medial interlaminar nucleus has an independent projection to the suprasylvian gyrus could not be determined. GAREY (1965) also states that the LGN projects ipsilaterally to areas 17, 18 and 19, but it is not clear whether the medial interlaminar nucleus is included in this statement.

No evidence was found of a contralateral cortical projection from the LGN in 11 cats and 2 monkeys. The contrary result by GLICKSTEIN, MILLER & SMITH (1964) is perhaps attributable to their approach through the corpus callosum damaging transcallosal fibres interconnecting the visual areas of cortex. As the single control lesion reported by these authors was ventral to the LGN, it is probable that the track was anterior to that used in the other experiments, and might thus have missed the forward edge of the relevant transcallosal fibres.

RESULTS - CORTICAL LESIONS

The purpose of these experiments was to provide more information about the nerve pathways emanating from area 17, linking adjacent areas and crossing the corpus callosum.

Representative coronal sections through the lesions have been reproduced in Figs. 20 - 21. The ventricles and the lesions are shown solid black. The dots give some idea of the density of preterminal degeneration but are not intended to give information about the depth within the cortex. If the degeneration in more anterior or posterior sections differs from the figure, this is mentioned in the text.

For convenience, the sites of most of the lesions have been marked on a single dorsal view of the cat brain, Fig. 19. The dotted line marks the approximate position of the area 17/18 boundary of OTSUKA & HASSLER (1962), which corresponds to the Visual I/II boundary of the electrophysiologists, BILGE et al. (1963) and HUBEL & WIESEL (1965). Lesions have been classified as :-

1. Unquestionalby within Visual I.
2. Those which could have been either in Visual I or II.
3. Those which could have been either in Visual II or III.
4. Lesions of the middle suprasylvian gyrus.

The point of division for groups 2 and 3 is an imaginary line through the centre of area 18 of OTSUKA & HASSLER (1965). A further point is that the lesions are situated in such a manner that these in group 2 are situated close to the representation of the vertical meridian and those in group 3 are approximately along the horizontal meridian.

It will be convenient to refer to the map of the visual field as it is seen on the cortex (WHITTERIDGE, 1966), see Fig. 6.

Degeneration in the posterior and middle blocks (i.e. the posterior 2.3 cm of brain) will be considered first.

Lesions within Visual I

Cat A

The lesion was in the splenial gyrus, commencing 8 mm anterior to the occipital pole and extending for about 5 mm. The lesion reached the white matter for only part of this distance. There was no sign of other cortical damage in either hemisphere.

Survival period : 11 days

Ipsilateral fibre degeneration was found in four areas. A moderate amount was found in the cortex around the lesion but most of the degenerating fibres entered the white matter. Another patch of degeneration was found on the medial half of the lateral gyrus starting 1 mm lateral to the mid-line. The third patch was dense in the medial wall of the accessory intralateral gyrus. There was a scant amount of degeneration on the lateral half of the middle suprasylvian gyrus.

Contralateral degeneration: a few degenerated fibres were found opposite the lesion at the topographically corresponding point. (Fig. 2$\frac{3}{2}$).

Cat B

The lesion was close to the region devoted to central vision, being 6 mm from the occipital pole, mid-way across the postero-lateral gyrus.

Survival period : 8 days

Ipsilateral degeneration: a moderate amount of fibre degeneration was found around the lesion. In the same coronal plane as the

lesion a scant amount of degeneration was found on the posterior end of the middle suprasylvian gyrus. The amount of degeneration increased 1 - 2 mm more anteriorly, where it was found in the bottom of both walls of the middle suprasylvian sulcus.

Contralateral degeneration : a moderate amount of preterminal degeneration was found at the point corresponding to the lesion. An occasional degenerated fibre was found on the medial half of the middle suprasylvian gyrus.

Cat C

The lesion was rather larger than in the previous cat and was situated more medially.

Survival period : 8 days

Ipsilateral degeneration was dense around the lesion and was moderate in amount in two areas of the middle suprasylvian gyrus, one medially and one laterally placed. There was slight degeneration in the medial wall of the middle ectosylvian gyrus, which merged with the lateral suprasylvian degeneration about 2 mm anterior to the lesion.

Contralateral degeneration was moderate in amount on the medial half of the lateral gyrus and slight on the medial half of the middle suprasylvian gyrus.

Cat D

The lesion was situated about 10 mm anterior to the occipital pole and about 2 mm below the dorsal surface on the medial wall of the hemisphere. The lesion did not penetrate the optic radiation.

Survival period : 14 days

Ipsilateral degeneration was dense around the lesion. Degenerating fibres could be seen to enter the white matter and also to run

through the grey matter parallel to its surface in roughly two bands, one at the upper and the other in the lower boundary of cortical layer IV. Amongst these fibres of passage, degenerating preterminals could be distinguished by their random distribution. This pattern of degeneration extended only a short distance inferior to the lesion but extended about a third of the way across the lateral gyrus. About two-thirds of the way across the lateral gyrus there was a small patch of degeneration which could be followed into the bottom of the lateral sulcus. There was a slight amount in the lateral wall of the middle suprasylvian gyrus. Contralateral degeneration was slight over the medial quarter of the lateral gyrus and occasional degenerated fibres were found opposite the lesion. A few degenerated fibres were found in the lateral wall of the middle suprasylvian gyrus.

Conclusion (See Fig. 21)

Degeneration ipsilateral to the lesion was found in three regions. The first was medially placed on the lateral gyrus. The second was found on the medial half of the intralateral accessory gyrus for the lesion some distance from the representation of the vertical meridian (cat A) but was on the medial wall of the suprasylvian gyrus for the lesions close to the vertical meridian (cats B & C). The third region of degeneration on the suprasylvian gyrus was less dense, tended to be situated a little anterior to the lesion and did not show a topographical relationship.

These scanty patches of degeneration are within Visual II, Visual III and SL as defined by the electrophysiologists. Visual III shows the expected topographical relationship. This is not apparent for the Visual II degeneration. The probable explanation

is that the lesions near the representation of the vertical meridian will also interfere with fibres running through the grey matter from Visual I in the upper medial wall of the hemisphere, to Visual II more laterally situated on the hemisphere, producing a greater extent of degeneration of Visual II than expected.

Contralateral degeneration was found at the corresponding point to the lesion, this was very scanty but definite for the lesion between the hemispheres. For the lesions near the representation of the vertical meridian, degeneration on the contralateral medial half of the lateral gyrus was moderate in amount. It was not possible to tell if this was in Visual I, Visual II or both. Also for the vertical meridian lesions a few degenerated fibres were found in Visual III contralaterally.

Confirmation of a projection to the medial wall of the hemisphere from a contralateral lesion comes from two other cats. These had additional damage; in one the lesion in the splenial gyrus extended into the white matter running to the lateral gyrus and in the other, the surface of the lateral gyrus was damaged.

Lesions in Visual I/Visual II

Cat E

The lesion was on the posterior end of the intralateral accessory gyrus, not quite reaching the white matter.

Survival : 8 days

Ipsilateral degeneration : most of the degenerated fibres from the lesion entered the white matter but a few extended laterally through the grey matter. A slight amount of fibre degeneration was seen on the medial wall and more anteriorly on the lateral wall of the middle suprasylvian gyrus.

Contralateral degeneration: occasional degenerated fibres were found on the medial extreme of the lateral gyrus, the intralateral accessory gyrus and the middle suprasylvian gyrus.

Cat F

The lesion was less lateral but more anterior than in the last cat. It reached the grey-white boundary.

Survival : 19 days

Ipsilateral degeneration was dense between the lesion and the medial edge of the lateral gyrus and a few fibres were found on the medial wall of the hemisphere. Although there were some fibres of passage lateral to the lesion only a few appeared to end near the lesion, A slight amount of degeneration was found at the bottom of the lateral sulcus and the lateral wall of the middle suprasylvian gyrus, particularly a little anterior to the lesion.

Contralateral degeneration: a slight amount was found opposite the lesion and it was scant on the middle suprasylvian gyrus.

Cat G

The lesion was a pinprick about 13 mm anterior to the occipital pole.

Survival : 22 days

Ipsilateral degeneration was dense around the lesion, radiating medially through the grey and cutting directly through the white matter to reach as far as the suprasplenial sulcus. Laterally many fibres of passage ran through the grey matter of the lateral gyrus to the medial wall of the lateral sulcus. Degenerated fibres which appeared to have a preterminal distribution were scattered along the lateral gyrus. There was dense degeneration across the whole of the middle suprasylvian gyrus, although on the crown it was composed chiefly of fibres of passage. A few fibres

were found on the medial edge of the middle ectosylvian gyrus. Contralateral degeneration was scant on the medial half of the lateral gyrus.

Cat H

This lesion was larger and situated slightly anterior to the last one.

Survival : 7 days

Ipsilateral fibre degeneration was dense around the lesion, extending in slight amounts as far as the suprasplenial sulcus. Across the lateral gyrus preterminal degeneration was slight, increasing in amount in the medial wall of the lateral sulcus. There was a moderate amount of degeneration in the lower part of the lateral wall of the middle suprasylvian gyrus. Contralateral degeneration was moderate in amount across the medial half of the lateral gyrus. The difference in the amount of degeneration compared with cat G is probably due to the difference in the size of the lesions.

Conclusion (See Fig. 22)

Particularly in the best stained sections degeneration could be seen down the medial wall of the hemisphere (Visual I). The lateral patches of degeneration in the region of the lateral sulcus probably correspond to Visual III. A third patch of degeneration was found in the lateral wall of the middle suprasylvian gyrus. Degeneration contralateral to the lesion could usually be found at the medial edge of the lateral gyrus in the region of the Visual I/Visual II boundary. This amounted to only an occasional fibre for the laterally placed lesion (which was likely to be a little distance from the vertical meridian) in cat E. The degeneration was more dense for lesions closer to the vertical

meridian (cats F, G and H). Very little was found in Visual III and the suprasylvian gyrus.

Lesions in Visual II/Visual III

Cat I

The lesion was on the lateral edge of the lateral gyrus penetrating to the junction between grey and white matter. The medial wall of the lateral sulcus was undamaged.

Survival : 7 days

Ipsilateral degeneration: a considerable number of degenerating fibres ran into the white matter from the lesion but there was little in the adjacent cortex. A few degenerated fibres were seen on the splenial gyrus. Fibre degeneration was dense on the lateral wall of the middle suprasylvian gyrus particularly more anteriorly.

Contralateral degeneration was scant opposite the lesion and on the middle suprasylvian gyrus.

Cat J

The lesion was the most anterior one of the series. It was on the lateral edge of the lateral gyrus but at the grey-white boundary it extended more medially than in the last cat.

Survival : 7 days

Ipsilateral degeneration: a slight amount of fibre degeneration was seen in the bottom and upper border of the splenial sulcus. Most of the degeneration around the lesion ran through the grey matter, around the bottom of the sulcus to a moderately dense patch of degeneration on the medial wall of the middle suprasylvian gyrus. Degeneration was dense in the lateral wall of this gyrus. Contralateral degeneration amounted to only an occasional degenerated fibre opposite the lesion and a scant amount in the medial

wall of the suprasylvian gyrus.

Conclusion (See Fig. 22)

Degeneration found in the medial wall of the hemisphere (Visual I) is consistent with the mapping of WHITTERIDGE (Personal communication). Lesion J was placed close to the horizontal meridian towards the periphery of the field. The degeneration was found in the bottom of the splenial sulcus which represents the region near the horizontal meridian at the periphery. Lesion I was less peripherally placed near the horizontal meridian and the degeneration in Visual I was less peripherally placed on the splenial gyrus.

As the lesions occur in the region of Visual II/Visual III boundary, they can give little information about a projection from Visual II to Visual III. In cat J, the degeneration in the medial wall of the suprasylvian gyrus can be interpreted as the Visual III degeneration due to the medial edge of the lesion damaging fibres from Visual II or Visual I close to the representation of the vertical meridian. The chief degeneration was found on the lateral wall of the suprasylvian gyrus.

Middle suprasylvian gyrus lesions

Cat K

The lesion was tiny at the posterior end of the middle suprasylvian gyrus, on its crown.

Survival : 7 days

Ipsilateral degeneration was present in small amounts only around the lesion.

Contralateral degeneration was not found, (but see discussion for significance of a negative finding).

Cat L

The lesion was larger and a couple of millimetres more anterior to

the last.

Survival : 7 days

Ipsilateral degeneration was found in moderate amounts around the lesion. A moderate amount of fibre degeneration was also found at the anterior end of the middle suprasylvian gyrus. A slight amount was found on the medial edge of the middle ectosylvian gyrus and an occasional degenerated fibre was seen on the splenial gyrus. Contralateral degeneration was found in small amounts at the posterior end of the middle suprasylvian gyrus.

Cat M

This lesion was more anterior and medial than in the previous animals. It was small but reached the surface of the white matter.

Survival : 7 days

Ipsilateral degeneration was slight around the lesion where it was found predominantly on the medial side running a short distance down the medial wall of the gyrus. A moderately dense area of degenerated fibres was found on the lateral half of the lateral gyrus, particularly in the lateral sulcus. This extended for about 4 mm along the gyrus on either side of the coronal level of the lesion. Posteriorly a small patch of degeneration was found on the crown of the splenial gyrus.

Contralateral degeneration - a moderate amount of fibre degeneration was found in the middle third of the lateral gyrus and an occasional degenerated fibre was found on the middle suprasylvian gyrus.

Cat N

The lesion was small and situated on the lateral edge of the crown of the middle suprasylvian gyrus about midway along its length. Ipsilateral degeneration spread out from either side of the lesion

across the whole of the gyrus. An occasional degenerated fibre was found on the medial edge of the ectosylvian gyrus. Some fine degenerated fibres were found in the bottom of the splenial sulcus and cingulate gyrus. This is in accord with CRAGG (1965). Contralateral degeneration - an occasional degenerated fibre was found on the middle suprasylvian gyrus.

Cat O

The lesion was small and more medially placed than the last although it occupied the same position along the length of the gyrus. Ipsilateral degeneration was found only locally around the lesion, and for a few millimetres anterior and posterior to it. Contralateral degeneration was not found.

Conclusion

Most of the degenerated fibres were seen to enter the white matter although there was some fibre degeneration in the grey matter on either side of the lesions. Two of the lesions on the posterior end of the middle suprasylvian gyrus produced occasional degenerated fibres in area 17 at a more posterior coronal level. Two animals were unusual; Cat L had a projection to the anterior end of the middle SUPRASYLVIAN gyrus and Cat M had a projection to the lateral gyrus. These findings might be attributed to differences in the size of the lesion, quality of staining or the topographical organization of the projection from the middle suprasylvian gyrus. They were not due to cortical damage. Little contralateral damage was found.

Layers of Termination

Because of the smallness of the lesions and the sparseness of

across the whole of the gyrus. An occasional degenerated fibre was found on the medial edge of the ectosylvian gyrus. Some fine degenerated fibres were found in the bottom of the splenial sulcus and cingulate gyrus. This is in accord with CRAGG (1965). Contralateral degeneration - an occasional degenerated fibre was found on the middle suprasylvian gyrus.

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Most of the degenerated fibres were seen to enter the white matter although there was some fibre degeneration in the grey matter on either side of the lesions. Two of the lesions on the posterior end of the middle suprasylvian gyrus produced occasional degenerated fibres in area 17 at a more posterior coronal level. Two animals were unusual; Cat L had a projection to the anterior end of the middle SUPRASYLVIAN gyrus and Cat M had a projection to the lateral gyrus. These findings might be attributed to differences in the size of the lesion, quality of staining or the topographical organization of the projection from the middle suprasylvian gyrus. They were not due to cortical damage. Little contralateral damage was found.

Layers of Termination

Because of the smallness of the lesions and the sparseness of

some of the projections the study was not well suited to reveal information on this point. Furthermore it is difficult to set a deep level to the degenerated preterminals because this is obscured by fibres of passage. Nevertheless a few conclusions can be drawn. Degeneration was more widespread than in the study of the LGN projection and degenerated fibres extended throughout the whole of lamina III. The pattern in terms of depth within the cortex did not appear to differ at the sites in which degeneration was found. In one cat (which had a dense projection to the contralateral gyrus), the degeneration extended well into layer II.

Anterior Blocks

These were studied in five of the previously described brains which had lesions on the lateral gyrus (cats C,F,G,H & J). No degeneration was found in four of the cats with the more medial lesions (cats C,F,G, & H). A few degenerated fibres were found on the anterior sigmoid gyrus ipsilateral to the lesion in cat J. A fifth cat P, had a larger lesion extending about 10 mm along the lateral margin of the lateral gyrus. A dense projection was found to the antero-lateral edge of the anterior sigmoid gyrus ipsilateral to the lesion and to the anterior end of the lateral gyrus bilaterally. Unfortunately there was a considerable amount of artefact in this brain.

As these small lesions are rather unsatisfactory for tracing degeneration which might be scant, the opportunity was taken to examine sections prepared by CRAGG (1965). In that study he was only concerned with the projection to the allocortex from bilateral lesions of the middle suprasylvian gyrus. Two brains were examined and fibre degeneration was found in the anterior sigmoid gyrus.

Fig. 19. Composite dorsal view of a cat cerebral hemisphere showing the positions of the various lesions.

Figs. 20 & 21. Coronal sections through the lesions shown in fig. 19, see text for details.

Fig. 22. A summary diagram to show the distribution of cortical fibre degeneration.

(a) From lesions within visual I.

(b) From lesions within visual I or II.

(c) From lesions within visual II or III.

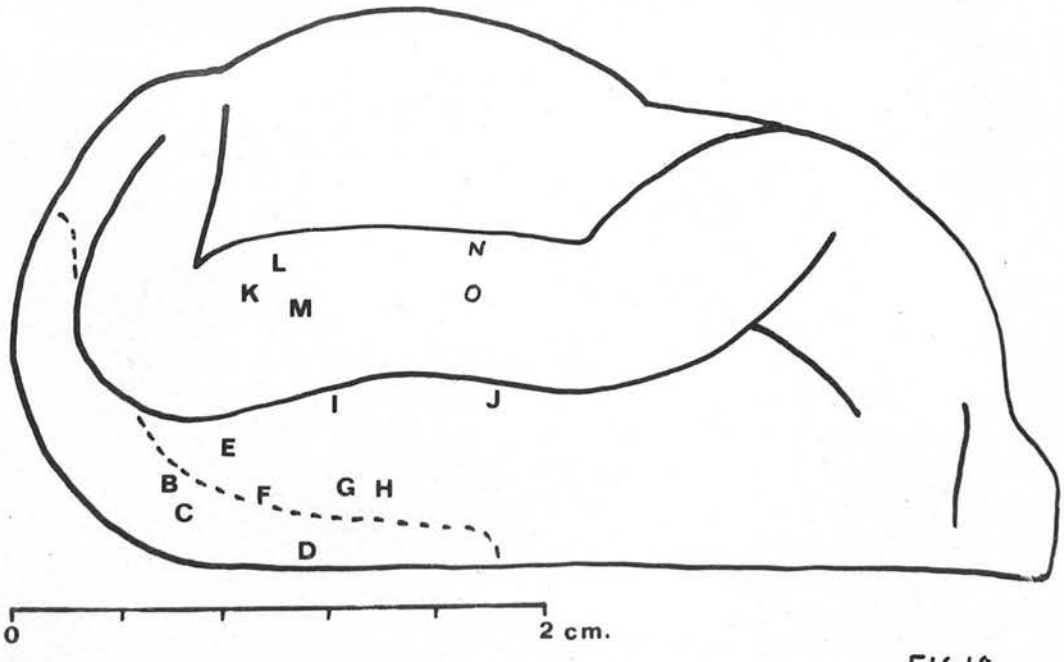


FIG.19.

FIG.20.

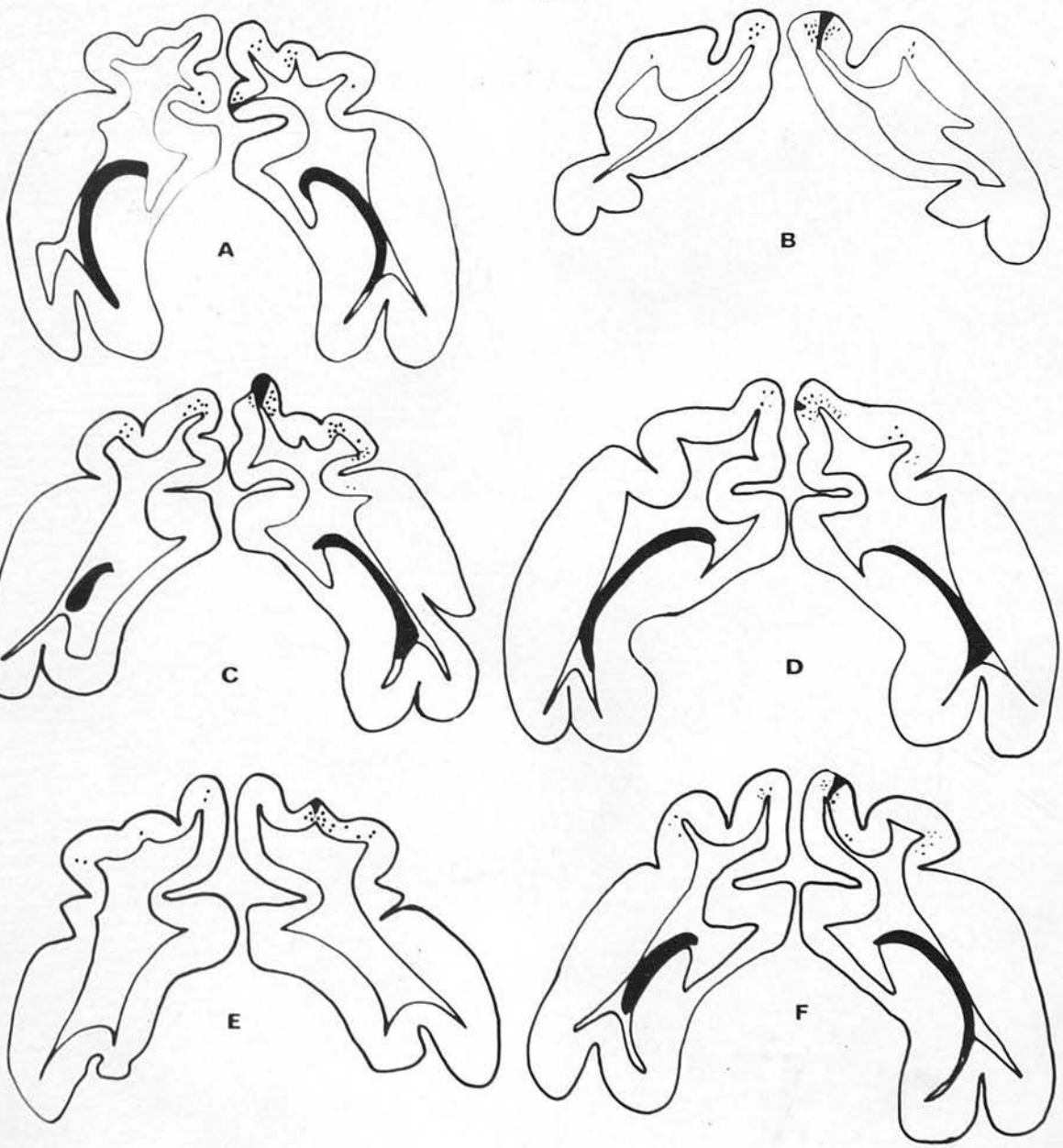
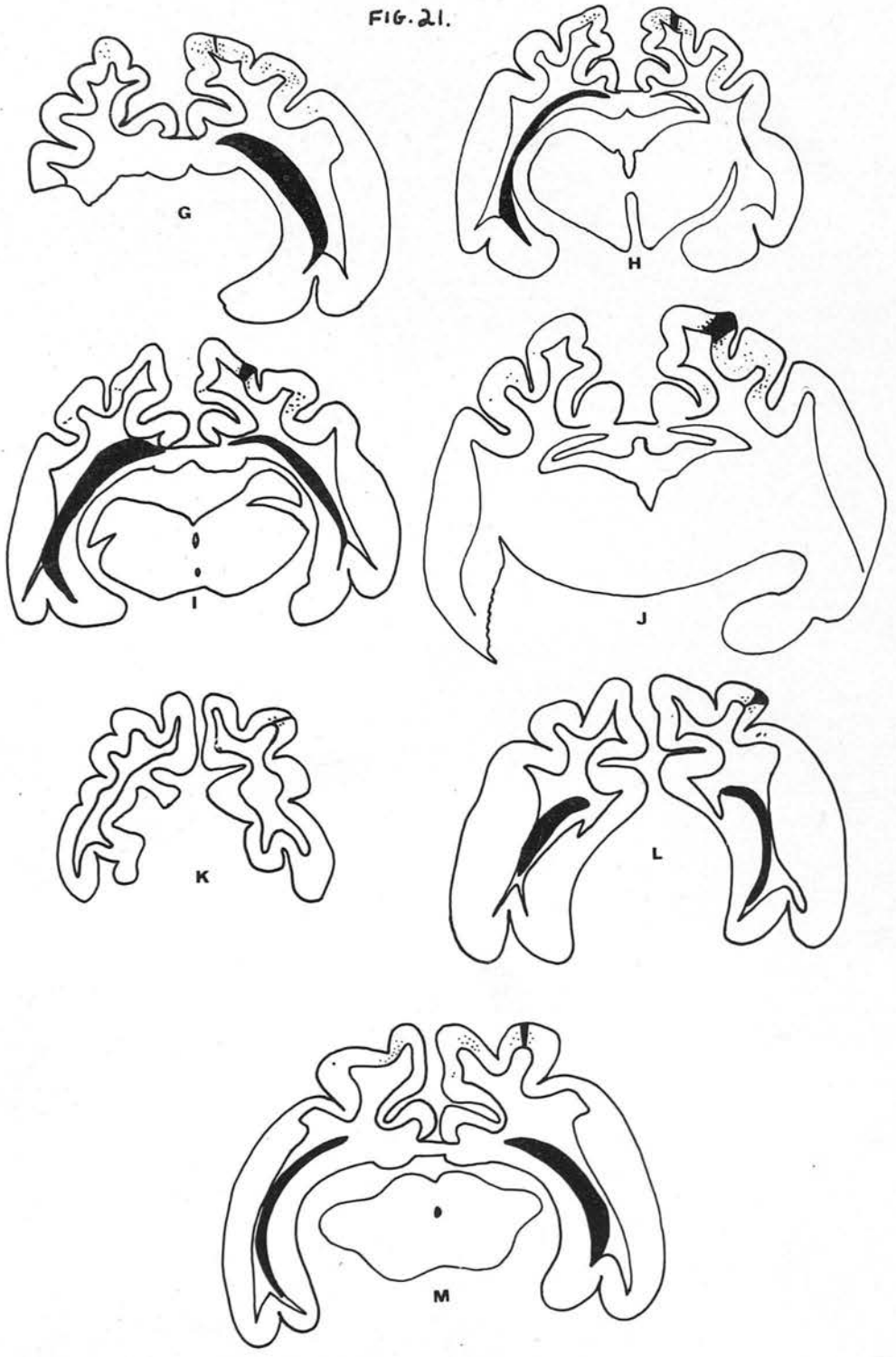


FIG. 21.



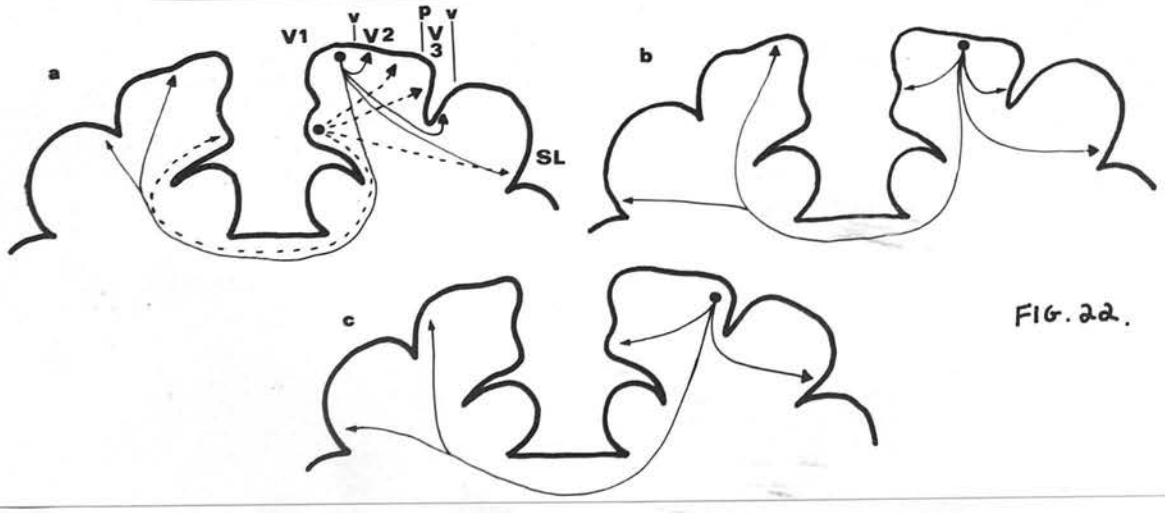
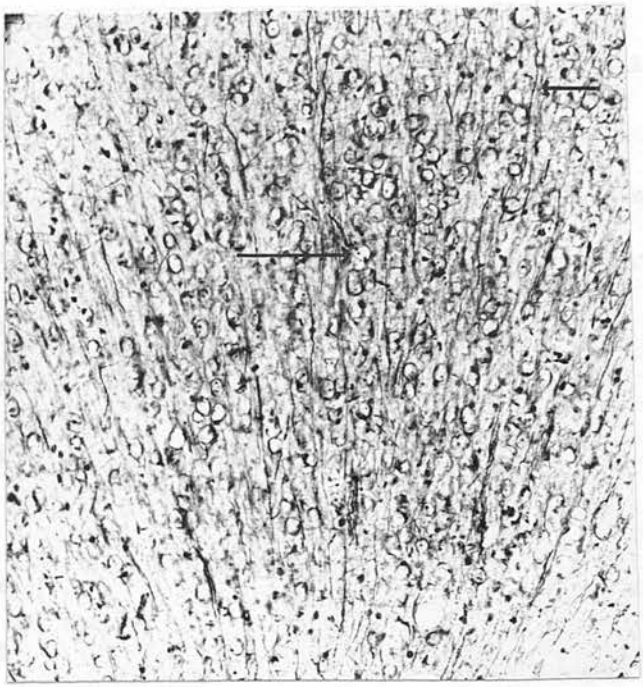


FIG. 22.

Fig. 23. A photograph of Nauta degeneration, marked by arrows, in the splenial gyrus contralateral to the lesion in cat A.



DISCUSSION

Cortical Lesions

Some of the problems found with the Nauta method have already been discussed. The following two points are more relevant to the cortical study.

1. The size of the lesion is a factor. A small lesion was excellent for topographic localisation but was not good for showing a sparse projection which might be missed. This could be avoided by using a large lesion but then there was the problem that the large lesion might extend into extraneous areas. It must be remembered that the lateral gyrus is only 4 - 6 mm wide and contains three visual areas.

2. It may be argued that some of the degeneration seen is retrograde. So far this interpretation has only been put forward in a few sites: the cat dorso-medial thalamic nucleus (GUILLERY, 1959); the rabbit LGN (CRAGG, 1962) and in the rabbit anterior thalamic nuclei (POWELL & COWAN, 1964). They describe a fine, regular, diffuse granular deposit which does not show any pericellular relationship ten days post-operatively when the retrograde Nissl changes are becoming obvious. CRAGG (1962) has reported the Nauta changes rather earlier at eight days in the rabbit but he does not comment on the Nissl appearance. At fourteen days according to POWELL & COWAN (1964) the granules become coarser and blacker and some coarse fibres start to break up. The typical anterograde Nauta degeneration picture occurs much earlier, and is fully developed at seven days (the pigeon optic nerve fibres being an exception) EVANS & HAMLYN (1956). This appearance of retrograde

Nauta degeneration is so different from the anterograde picture, that thetwo are unlikely to be mistaken, particularly if short survival periods are used. GRANT (1965) has described in a brief communication that after cutting the hypoglossal nerve the dendrites of the cell bodies broke up and could be mistaken for anterograde axonal Nauta degeneration. However, this may be exceptional since the animal used was an eleven day old kitten. This phenomenon has not yet been described in the adult cat.

The cortical degeneration described in the present series of experiments had all the characteristics of orthodox anterograde Nauta degeneration and survival times were often only seven days. However, the degeneration appeared the same even when survival periods of up to three weeks were used.

The finding of three patches of degeneration ipsilateral to an area 17 lesion has confirmed the work of HUBEL & WIESEL (1965). The first patch was found on the middle third of the medial-lateral extent of the lateral gyrus.

The second patch of degeneration was found in the medial wall of the lateral sulcus with a lesion near the horizontal meridian, and in the lateral wall for a lesion close to the vertical meridian. This area then is a mirror-image about the horizontal meridian of the previously described Visual II and corresponds to Visual III of the electrophysiologists. The lateral extreme of Visual III which corresponds to the vertical meridian is found on the medial side of the middle suprasylvian gyrus, in the mouth of the sulcus. This is a good deal more lateral than the Fig. 34 of HUBEL & WIESEL (1965) (Fig. 8) would suggest, but it has already been pointed out that their finding is inconsistent with their physiological findings of Visual III being a mirror image of Visual II joined along a line representing the peripheral visual field. It is consistent with their figures for more posterior regions.

The third patch of degeneration on the lateral edge of the middle suprasylvian gyrus does not appear to be topographically organised.

The more posterior lesions in Visual I (corresponding to central vision) projected a few millimetres rostrally to the middle suprasylvian gyrus. This confirms the observation by CLARE & BISHOP, G.H. (1954).

The projections are not strictly point-to-point but extend for one or two millimetres, gradually becoming less dense, on either side of the centre of the patch of degeneration. The Visual III degeneration is less discrete than Visual II, (see also p.96)

More laterally placed lesions on the lateral gyrus project predominantly to the lateral wall of the suprasylvian gyrus.

Area 17 receives some fibres from more laterally situated areas. This is quite definite for lesions close to the Visual I/II boundary which probably also damage fibres running from more lateral regions of Visual II (i.e. peripheral visual field) to the representation of the peripheral field of Visual I. There is also a projection from the lateral edge of the lateral gyrus, linking areas close to the horizontal meridian, and from the posterior end of the middle suprasylvian gyrus.

Degeneration from a lesion of the middle suprasylvian gyrus is found in adjacent regions of the gyrus and further forward in the anterior sigmoid gyrus. This latter finding may be of great importance in linking the visual and motor centres. It confirms the electrophysiological findings of IMBERT et al. (1966). A projection to the splenial sulcus and the cingulate gyrus from a lesion half way along the length but not from more posterior regions of the middle suprasylvian gyrus confirmed CRAGG (1965). The results also suggested that the projection arose from the

lateral half and not the crown of the gyrus. A medially situated lesion on the middle suprasylvian gyrus was found to project bilaterally to the lateral gyrus whereas the other lesions did not. These findings of a differential projection of the medial and lateral halves of the gyrus need further confirmation.

Similarly, larger lesions are required to confirm whether Visual III but not Visual I and II projects to the anterior sigmoid gyrus.

It will be more convenient to discuss the contralateral degeneration with the electrophysiological findings.

ELECTROPHYSIOLOGICAL INVESTIGATIONS

The following experiments were undertaken to discover more about the properties of the neurones which link the visual areas of the two hemispheres.

Whereas the anatomical investigations only show the connections in relation to the gyral pattern of the brain, the electrophysiological investigations show in addition that the neurones have receptive fields close to the midline and the characteristics of their receptive field could be defined.

Methods

Ten cats were anaesthetized by placing them in a glass-fronted, air-tight cage into which a trilene and air mixture was pumped. This produced a smooth and relatively quick anaesthesia without excitement. Anaesthesia was then maintained with 75 mg. chloralose/kg. After ten hours, a further dose of chloralose was sometimes necessary.

A polyethylene cannula was inserted into a vein and in later experiments an artery to give a continuous record of blood pressure. A tracheal cannula was inserted. Body temperature was maintained with a thermostatically controlled electric blanket.

The left optic tract was cut just behind the chiasm, by an approach through the mouth. A Zeiss binocular operation microscope with 9 inch working distance was used. The jaws of the cat were held apart by two horizontal rods. The soft palate was cut longitudinally in the mid-line for about ½ inch just behind the margin of the hard palate. Packs were inserted to stop the bleeding and the margins of the wound were then held apart by stay sutures. A hole was drilled through the basisphenoid bone with a dental drill, initially in the mid-line until the chiasm could be seen and then it was extended postero-laterally over the left optic tract. The dura was opened using

specially shaped instruments. The pia over the tract was cut and the tract sectioned using a blunt hook. Haemostasis was secured with small packs which were then removed. The dural defect was covered with a small piece of polyethylene sheet and the hole in the bone plugged with 'Coe-pak', a periodontal dressing. 10 - 20 ml Dextran was given to replace lost blood.

Three animals were used for long term behavioural experiments. These were anaesthetized with sodium pentobarbitone ('Nembutal') 0.5 ml/kg body weight, intra-peritonealy. Full aseptic precautions were taken. Antibiotics were not given. Healing was prompt. In the terminal experiment they were anaesthetized with trilene and chloralose and were dealt with as the rest of the animals.

A rectangular hole, approximately 1.5 x 2.0 cm, was cut in the skull, centred over the sagittal sinus. The dura was removed as closely as possible up to the sinus. The hole was filled with 4% agar containing a little gelatin at 42°C.

The head was fixed by ear clamps and a jaw-piece in a stereotaxic machine specially designed to allow a full field of vision. The Clarke-Horsley plane was levelled with a spirit level.

The eye was tied to a brass ring with four sutures through the limbus and rotated until the pupil was vertical. The pupils were dilated with atropine and the fundus was observed by an indirect ophthalmoscope and a mirror placed at the centre of the perimeter. The brass ring was moved until the image of the area centralis occupied the centre of the perimeter. In early experiments a plain contact lens was placed over the eye, but in later ones the eye was refracted and a correction lens inserted.

The position of the stimulus was located on an Aimark perimeter whose arm had a diameter of 33 cm. In the darkness, a neon tube 5 mm in diameter which flashed for about 5 m.sec onee a second,

triggered by the sweep of the oscilloscope, was used to search for responses. An error of 1 cm in locating the centre of Unit's field on the perimeter is equivalent to an error of 2° of visual angle. When a unit response was found it was studied in more detail using black bars or edges against the illuminated light grey of the perimeter. Alternatively a bright slit of variable dimensions was projected onto the perimeter in darkness.

Recordings were made using tungsten microelectrodes as described by HUBEL (1959).

In most experiments the Visual I/Visual II boundary was cooled on the right side of the cortex with a block of enamelled copper about 5 mm x 3 mm. Alcohol which had been cooled by a salt and ice mixture circulated through the block. In later experiments a needle cooled by the expansion of propane (DONDEY, ALBE-FESSARD & Le BEAU, 1962) was used. This had a small metal foot to increase the area resting on the cortex. A tiny thermode on the foot allowed the temperature to be measured. This was controlled between 0 - 10°C during cooling.

At the end of the experiment the cat was killed with an overdose of sodium pentobarbitone and was perfused with formol saline. A small block of brain containing the optic chiasm was removed and embedded in low-viscosity nitrocellulose. The region of the cut tract was closely scrutinized during section cutting. Every fifth section, 25 μ thick, was stained by the Weil* method.

The posterior part of the brain was cut on a freezing microtome at 50 μ and every section stained with cresyl violet. The sections were enlarged three times with a photographic enlarger and the outline traced to show the position of the needle track. Most of the electrode insertions were short but nevertheless, the tracks could usually

* See Appendix

be found, and the rest could be interpolated. Occasional marking lesions were made during the experiment by passing current through the recording electrode.

Results

Some of these findings have already been published, CHOUDHURY, WHITTERIDGE & WILSON, 1965.

Firstly, the right hemisphere was explored to determine the vertical meridian. This also served as a check on the state of the animal and the electrode. At any stage in the experiment if these were in doubt, the response was checked on the right.

The left hemisphere was then explored usually from the mid-line in 1 mm steps working laterally. Other rows were then placed in 1 mm steps anterior or posterior to the first row.

Responses were predominantly found 3 - 4 mm lateral to the mid-line (Fig. 25). Comparison with the right hemisphere showed that this region covered the vertical meridian of the visual field. A stimulus through either eye was effective. No responses were found 1 mm lateral to the mid-line. In the whole series of experiments only three needle stabs showed responses at 2 mm from the mid-line. This medial region of the lateral gyrus was explored intensively. The needle was inserted to a depth of 6 - 7 mm so that the peripheral visual field in the splenial sulcus was also investigated. In explorations anterior to the stereotaxic plane A1 responses were not found lateral to the active strip although this was not examined systematically beyond 5 or 6 mm lateral to the mid-line. Late responses were observed in the middle suprasylvian gyrus but these were not studied in detail. Between A1 and the area centralis region (P4) responses were found up to 8 mm laterally. This presumably corresponded to the Visual III meridian (Fig. 26).

The centres of the receptive fields of the penetrations shown in Fig. 25 were plotted in Fig. 27, and were all found to be close to the vertical meridian. The receptive fields of the penetrations of Fig. 26 were all within 4° of the centre of the visual field.

Eighteen single units were recorded. Their receptive fields were found to be band-shaped when examined with slits of light, black bars of different dimensions or black edges. Movement always evoked a brisk response which was asymmetrical in some units. With one exception, units were sharply localised and could be classified as 'simple units' (HUBEL & WIESEL, 1962). Units had receptive fields orientated in all possible directions although only one example was found with a vertical orientation (i.e. it required horizontal movement to elicit a response).

Responses in the active strip around the vertical meridian on the left side were reversibly abolished by cooling the corresponding point in the right hemisphere below 10°C. Cooling an area 3 mm in front of the corresponding point was ineffective showing that the neuronal origin in the right hemisphere of the response was localised.

The responses from four laterally placed units with fields in the centre of the visual field could not be abolished by cooling for 4 - 5 mins. at the Visual I/Visual II boundary contralaterally.

A response was once reversibly abolished by lowering the foot of the propane cooled needle onto the splenium of the corpus callosum. A series of electrolytic lesions was made in the corpus callosum, one of these temporarily abolished the response and another had a permanent effect. At the end of another experiment the corpus callosum was cut by a knife inserted between the hemispheres. Responses could still be obtained from the right hemisphere but were now completely absent on the left.

Latencies were variable, between 25 - 35 msec. to small flashes of light on the perimeter. No clear difference was detectable between the hemispheres. Increasing the flash repetition rate from 1/sec to 3 - 4/sec had no effect on the normal hemisphere but sometimes caused a decay in the responses recorded on the left.

It is interesting to note that visual responses were still found in the hemispheres of two cats which had had no direct visual input for over two years, and in another cat for seven months.

Conclusion

After cutting one optic tract early visual responses could only be found on that side in the region of the vertical meridian and central area of the visual field represented upon the cortex. The responses from points on the vertical meridian could be abolished by cooling the corresponding point in the other hemisphere or by cutting the corpus callosum. Responses in the central area could not be abolished by cooling, presumably because the area with callosal connections was now greater than the narrow strip which could be cooled. The Visual III response was probably not abolished by cooling because the cooler was on the contralateral Visual I/Visual II vertical meridian and the area centralis response was not abolished because of the large area with callosal connection.

Fig. 24. Tracings of coronal sections from the cat used to construct fig. 26 at the stereotaxic levels indicated. The solid black lines show the positions where needle tracks could be found, the dotted lines estimated positions.

Fig. 25. A plot of electrode positions on the left and right hemispheres . IAP - intra-aural plane. Ordinate - stereotaxic planes, abscissa mm. to left or right of the midline. These points had receptive fields indicated in fig 27. O - no response. + - response in left hemisphere ● - response in right hemisphere.

Fig. 26. Left hemisphere of a different cat plotted as in fig. 25. O - no response. N - responses recorded but not abolished, and A - responses recorded and abolished by cooling the contralateral visual I/II boundary.

Fig.27. The position of the receptive fields of the points plotted in Fig. 25. O - receptive fields of points in normal right hemisphere. ● - receptive fields obtained from left hemisphere. The broken line indicates the vertical meridian of the right eye. There had been an error of about 10° in its fixation.

(Figs. 25 & 27 previously reproduced in CHOUDHURY, B.P., WHITTERIDGE D. & WILSON M.E. 1965.)

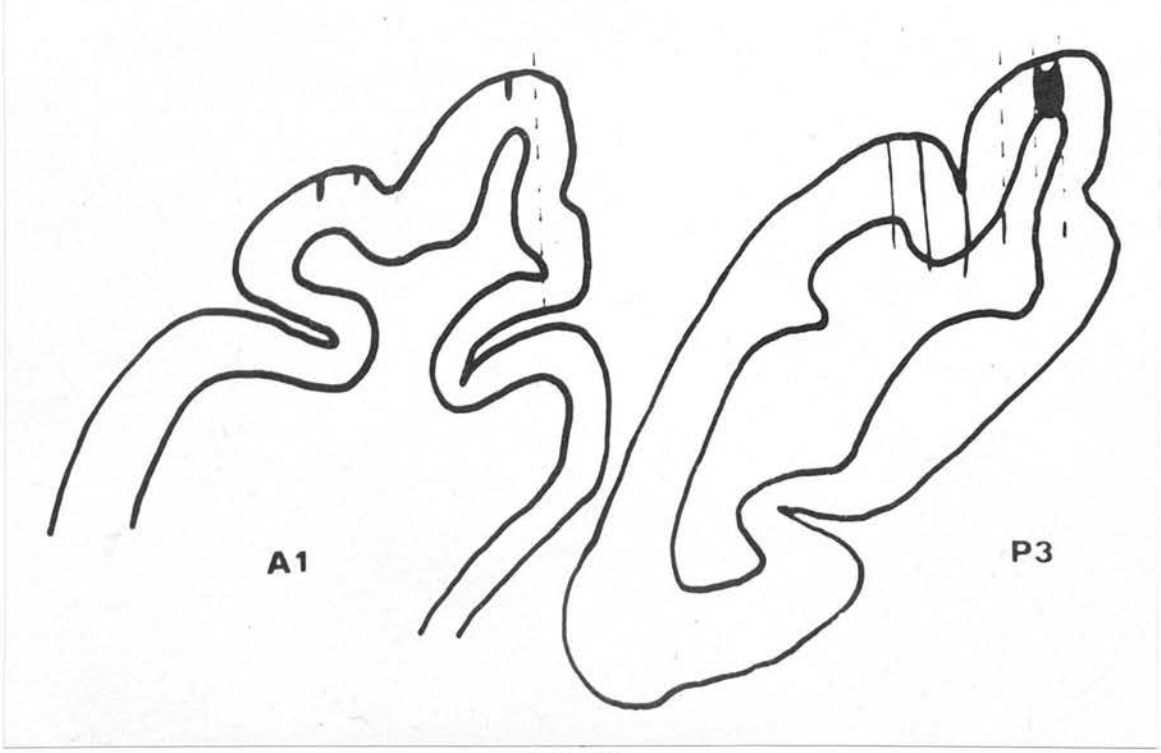


FIG. 24

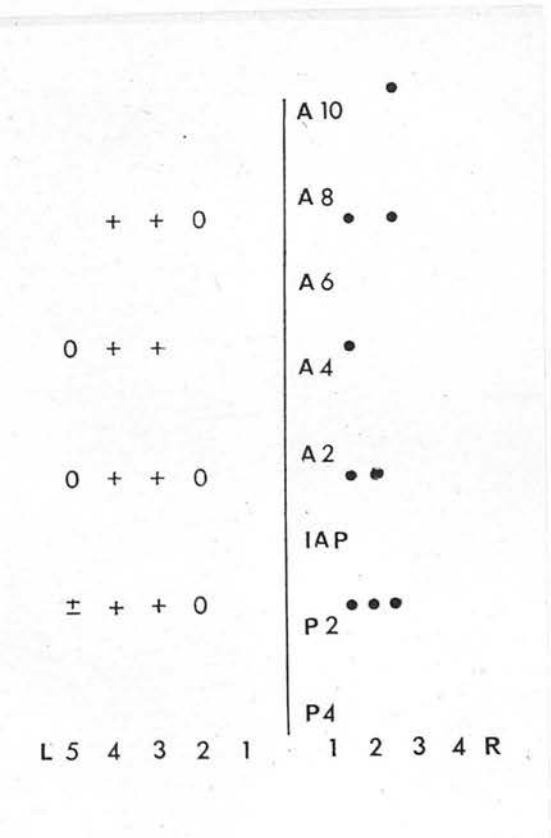


FIG. 25.

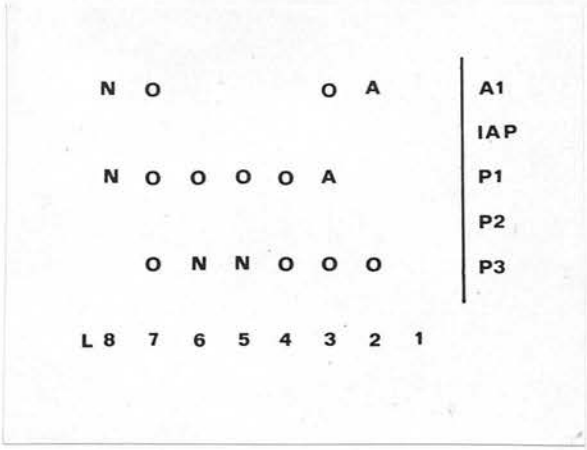


FIG. 26.

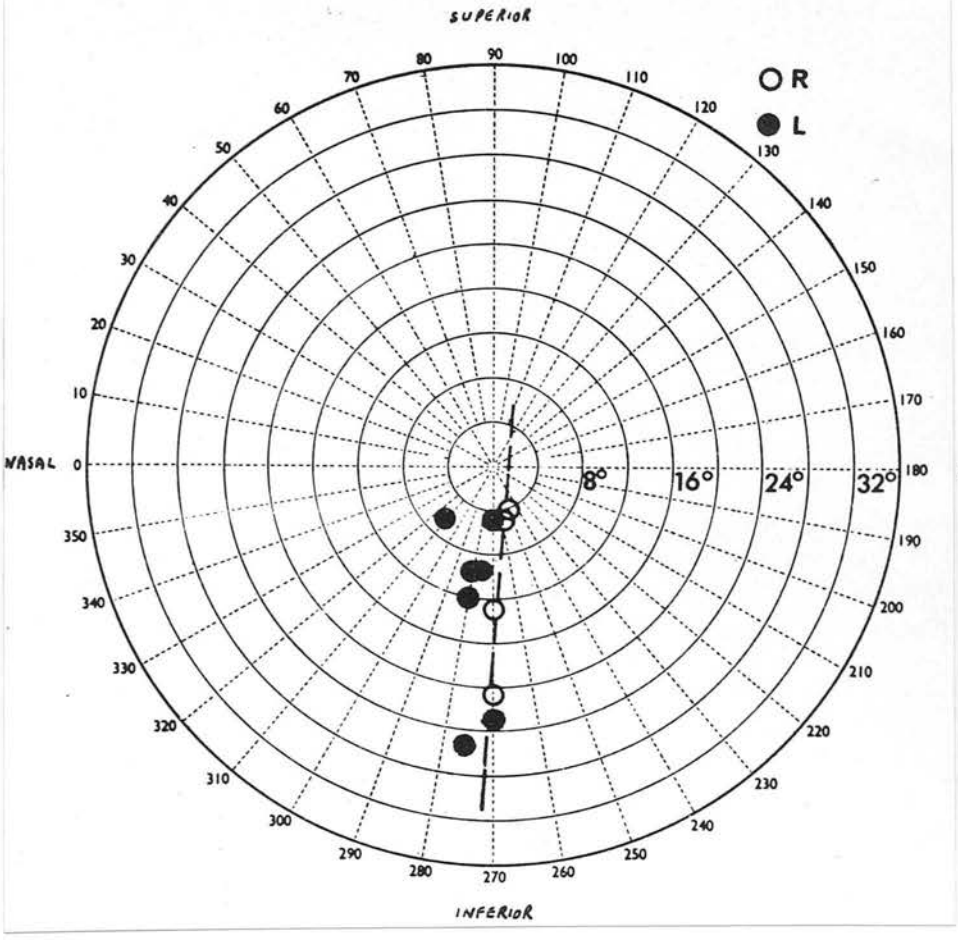

SUPERIOR
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FIG. 27.

DISCUSSION

Callosal Connections

The anatomical findings indicate a projection from area 17 to the contralateral areas 17, 18 and 19, but not to the lateral part of the suprasylvian gyrus. In one animal HUBEL & WIESEL (1965) found degeneration in the latter site. A scant projection may have been missed in the present experiments because such small lesions were made.

The densest degeneration was found on the medial half of the lateral gyrus; medially this extended up to the medial edge of the hemisphere and sometimes a little below the dorsal surface on the medial wall of the hemisphere. This is in agreement with the cross-sections of Nauta degeneration published by EBNER & MYERS (1965) and HUBEL & WIESEL (1965). This medial edge is well within area 17 on the map of OTSUKA & HASSLER (1962) and within Visual I of the electrophysiologists BILGE et al. (1963) and HUBEL & WIESEL (1965).

Contrary to EBNER & MYERS (1965) a slight projection was found to the medial wall of the hemisphere i.e. peripheral visual field. Part of this projection is due to 17 - 17 connections. This degeneration is only slight and in the presence of dense degeneration in the rest of the brain it is not surprising that it was missed by EBNER & MYERS (1965). It confirms the findings of POLLEY & DIRKES (1963).

EBNER & MYERS (1965) (Fig. 9), described three other areas which received few connections from the other hemisphere; the lateral part of the lateral gyrus about midway along its length; the postero-medial edge of the middle suprasylvian gyrus; and the lateral part of the middle suprasylvian gyrus about midway along its length. In the present experiments degeneration was found in these areas although

it amounted to no more than a few fibres.

This study complements the one of EBNER & MYERS (1965) who saw the total degeneration picture after removing one hemisphere. This gives no idea of the source of the various projections. The present method gives this information but because the degeneration is often slight and there is variability in the size of the lesion it is less reliable about the relative density of termination of the projections.

In contrast to the diffuse contralateral projections seen with the Nauta method the electrophysiological results indicate a strip a couple of millimetres broad lying on the vertical meridian on the dorsal surface of the lateral gyrus. Although there was no systematic electrophysiological mapping beyond 5 or 6 mm lateral to the mid-line of regions either superior or inferior to the centre of the visual field some evidence was found for a laterally located area on the medial half of the middle suprasylvian gyrus. More posteriorily callosal responses were also located less laterally. These latter responses were recorded from an area which seems to correspond to the area of degeneration which extends across the lateral gyrus (EBNER & MYERS, 1965) (Fig. 9). If these findings are extrapolated the result is a band with callosal connections running along the vertical meridian at the Visual I/Visual II boundary, fanning out at the area centralis across the lateral gyrus into another band corresponding with the Visual III vertical meridian.

Responses are recorded in regions of dense Nauta degeneration. It would appear that where the projection (as seen with the Nauta method) is sparse, this is insufficient to dominate the behaviour of a unit so that under these circumstances it appears unresponsive.

BEHAVIOURAL EXPERIMENTS

These will not be reported in any detail as the results are negative and only two cats were studied. It was originally planned as a pilot study and it did not seem worthwhile pursuing it further.

The aim of the experiment was to discover more about the function of the 'B' layer of the LGN by cutting the left optic tract and studying the animal's behaviour when wearing a monocular mask. Using the left eye the animal has an input to the A and B layers, whereas with the right eye it only goes to the A1 layer. Since the A and A1 layers have similar electrophysiological properties it seems reasonable to attribute any advantage shown with the left eye to the B layer. This makes the questionable assumption that non-geniculate fibres will not be concerned in the animals performance, and that the binocular overlap of the projection from the retina to the LGN is not significant.

SENEVIRATNE (1962) had shown that B layer units had a lower threshold to light and had larger receptive fields then the A or A1 layers. Most of the units fired at 'off'. This suggested that the most likely tests might be of threshold to light, the ability to distinguish objects which had blurred outlines and the differentiation of objects which differed in the darkness of their centres.

Methods

Operative details are given under electrophysiological methods, (p.118). One cat was trained before operation on a brightness discrimination problem. The other was naive. During the first post-operative week they were trained to wear a black cloth mask over one eye (MYERS, 1955). Training in the discrimination box began three

weeks post-operatively. The box measured 3' x 2½' x 2' and had a light-proof lid. At one end of the box a shutter could be raised to reveal two opal perspex hinged panels. Food cups loaded with 'Kit-e-Kat' cat meat were behind the panels. The panels could be bolted. Their movement was indicated by an extremely dim, shaded neon bulb which was operated by a sensitive switch. A non-correction procedure was adopted. Stimuli were presented according to the Gellerman series. As interest lay in the final level of performance and not in the rate of acquisition, the number of trials per day was not standardised. Blocks of twenty trials to each eye were given and then the mask was changed and another twenty trials were given. At first only forty trials a day were given, but as the animals became more sophisticated a hundred trials were common. Training was stopped as soon as there was any indication that performance was falling off. The order in which the eyes were tested was changed from day to day.

For threshold measurements a dark neutral density filter was placed in front of a 6v. bulb in a box with a diffusing opal perspex front. This was stood in an enclosed tunnel 4' from the stimulus panels. The stimulus panels were covered with black photographic paper except for a 2" diameter hole in their centre. Neutral density filters or an opaque plate could be fitted in front of these holes to produce the various stimuli. The stimulus used for the 'edgeless' brightness problem was built out of a series of pieces of tracing paper with holes of different diameters cut out of their centres. This 'edgeless' hole plus the various neutral density filters formed the positive stimulus, the negative was an opaque plate. The O and • were trans-illuminated in a similar manner.

The box was built as light-proof as possible and the room was in complete darkness. The cats were dark-adapted for half an hour before the experiment began and had not been fed since the previous day.

The intensity of light was measured and checked periodically at the stimulus panels using an SEI photometer. This was then reduced by a known amount by the Kodak 'Wratten' neutral density filters.

Results

Subsequent histology established that the optic tracts had been cut and that some additional damage had been inflicted deep to the tract.

The cats were handicapped by the lesion. A field defect could be demonstrated and they were impaired in their ability to walk along table edges. They had to be retrained to find the food in their feeding-cups and to overcome the neglect of the stimulus falling in their blind field. Inspite of this both cats became highly trained.

1. The absolute threshold for light was determined for one cat (9 months post-operatively) as 1.1×10^{-6} millilamberts (mL). There was no difference between the two eyes. A human volunteer had a threshold of 7.6×10^{-7} mL under the same conditions. A criterion of 80% correct was used to facilitate comparison with GUNTER. The human figure is about ten times that reported by GUNTER (1951), the cat figure is about ten times that of his worst cat and almost a hundred times greater than his average. There are a number of possible explanations which could account for this discrepancy but probably the most important is that Gunter used normal animals whereas this cat had a field defect so that it had to turn its head to compare stimuli and secondly it was wearing a monocular mask which may have been irritating. There were difficulties for the human too, he was locked inside the box on all fours peering at the stimulus panels at ground

level. Nevertheless, even though the extremely low threshold of illumination was not reached no difference was found at levels which were well in the scotopic range.

2. This experiment was repeated with the same cat using a positive stimulus which was an edgeless spot, light in the centre and dark at the periphery. This was made progressively darker by putting neutral density filters behind it. The negative stimulus was completely black. Again no difference was found between the eyes.

3. The other cat was trained to respond, eight months post-operatively, to the brighter of two lights. The difference was then reduced until the animal was unable to discriminate between them. No difference between the eyes was found when testing was stopped with positive stimulus 1.1×10^{-2} mL and the negative stimulus 1.7×10^{-3} mL (i.e. a difference of 0.8 log units.) To the human eye these stimuli were quite different but the cat had begun to behave erratically. In view of the difficult testing situation, this may well have been a motivational problem rather than an indication that the threshold was near.

4. This second cat was then trained to discriminate between transilluminated $\boxed{\text{O}}$ and $\boxed{\bullet}$ of equal area. Earlier attempts with both cats had failed but this time the negative stimulus was darkened using neutral density filters so that in the early stages the cat was making a brightness discrimination. The density was gradually diminished until the cat was left making a form discrimination. There was no significant difference between the eyes. The negative stimulus, the spot, was then cut out of

a neutral density filter so that its diameter equalled the outer diameter of the positive stimulus. The density of the filter was chosen so that both stimulus panels still transmitted the same amount of light. Performance was at chance level for both eyes.

Conclusion

No difference could be found between the eyes. None of the hypotheses tested could be substantiated. Either the wrong hypotheses were examined or, the assumptions ~~in the introduction (p.34)~~ were wrong. The results do not necessarily conflict with SENEVIRATNE'S finding that the 'B' layer cells had a lower threshold. The thresholds measured in electrophysiological experiments are well above those found in a behavioural situation (MARRIOT, MORRIS & PIRENNE, 1959). The factors which depress the response in the former situation (e.g. anaesthesia) may affect the different laminae differently.

PERSISTENCE OF CELLS WITHIN THE LGN AFTER WIDE REMOVAL OF VISUAL CORTEX

Whether neurones remain intact in the LGN after removal of the visual cortex is disputed (see p.29 of review). It seemed worthwhile repeating this work and at the same time looking to see if there was a difference in the laminar distribution of any remaining neurones.

Methods

Extensive neocortical ablations were made on one side in four cats to remove all possible cortical LGN projections. Three of the cats survived 7 - 9 weeks. The other had had the optic tract cut 2 years 4 months previously on the side on which the cortex was removed. At the second operation an attempt was made to make an incision medial and posterior to the LGN. The survival period was three weeks.

Histological procedures varied. Two were perfused with saline followed with formol-saline and sections were cut 30μ thick, one para-sagitally and the other coronally, on a freezing microtome. The other two were perfused with Heidenhain's Susa fixative, one was embedded in paraffin and the other was double embedded in celloidin and paraffin. Both were cut in the coronal plane, one at 50μ thick and the other at 30μ. Selected sections were stained with cresyl violet and with the Weil method.

Results

The whole of the projection of the LGN to the postero-lateral, lateral and middle suprasylvian gyri had been removed. A portion of the uncinate and dentate gyri and the most anterior few millimetres of frontal neocortex remained.

The attempt to isolate the LGN from more medial structures had

failed. Only the medial part of the pulvinar was destroyed and a small cut extended through the centre of the superior colliculus and became larger in the inferior colliculus.

In the LGN gliosis was severe, but except for the sections cut at 50μ lamination was still apparent. Normal neurones persisted in all laminae. These all measured under 18μ at their greatest diameter in laminae A and A1 and under 12μ for lamina B cells. This contrasts with the normal size range of 10 - 40μ for lamina A and A1 and 25μ for the B lamina.

Conclusion

Neurones do persist in the LGN inspite of the removal of the whole of its neocortical projections. The absence of large cells confirms FISCHMAN & MEIKLE (1965). The small cells remaining are likely to be the short-axon cells of O'LEARY (1940), or ones projecting to the sub-cortex. Unfortunately an attempt to destroy possible pathways to the superior colliculus and regions medial to the LGN failed. The marked degeneration within lamina B indicates a cortical projection from this lamina i.e. the whole of its projection does not go to the thalamus medial to the LGN (BISHOP & CLARE, 1955).

DISCUSSION

General

The receptive fields of the LGN cells are concentric whereas the cortical cells have elongated receptive fields. To achieve this HUBEL & WIESEL (1962) suggested that the cortical cells receive their input from a number of ganglion cells. It is therefore reasonable to find with the Nauta method that the preterminals of the LGN axons extend over a distance of cortex corresponding to at least a few degrees of visual field. This distance will be even greater for complex units than for simple ones. Consequently one finds an even greater spread in Visual II and Visual III. This is also likely to be the case for the formation of hyper-complex units from complex units.

It seems likely that the receptive fields of some units, particularly complex and hyper-complex ones, will cross the vertical meridian. Their component LGN neurones will then be in different halves of the brain, projecting to their own hemispheres. To complete these cortical units one would expect the existence of transcallosal neurones, to carry over information about the other part of the field of the unit. This would be likely to occur most frequently for units lying close to the vertical meridian but occasionally it would be necessary for the few units with large receptive fields to receive information many degrees away from the midline.

HUBEL & WIESEL (1965) have found a small overlap of the receptive fields at the vertical meridian, although they were unable to determine whether this was real or an artefact due to an error in lining up the eye. In the region of the projection of the area centralis it only amounts to a couple of degrees but this represents

one or two millimetres on the cortex. Therefore one might expect a projection from a point this far medial to the vertical meridian to project contralaterally.

Although they do not report receptive fields crossing the midline for more than a couple of degrees, a more extensive overlap may occur further from the central area, along the vertical meridian, where receptive fields are likely to be larger. This region was not studied by them in any detail. These units are probably rare and may have been missed. Finally, the boundaries of the receptive fields may not be as sharply defined as they suggest. More distant regions may influence the units although this effect may be too slight to be observed in their experimental situation. BURNS & PRITCHARD (1964) in the Cerveau Isole cat found much larger receptive fields in the central visual area than HUBEL & WEISEL.

In general the results presented confirm some of the findings of HUBEL & WIESEL (1965). Together they lend support to the idea of a projection from the LGN to the simple cells in Visual I and Visual II. In this way the pattern of the receptive field is transformed as complex cells in these regions receive projections from the simple cells and in hypercomplex units are formed from a combination of projections from the simple and complex cells. In this way features of the scene viewed by the eye are progressively abstracted, from the stage in the LGN where they are little more than a series of dots as in a newspaper picture, to the cells of Visual III which only respond to say a corner, appropriately orientated, but generalised over a large region of the retina. This transformation occurs as the visual information is passed laterally in the cortex from Visual I to Visual III. The appropriate cortico-cortical connections have been found and furthermore they maintain some degree of topographical localisation.

One might imagine that the next stage might be further abstraction in the lateral edge of the suprasylvian gyrus. This region has not been studied by HUBEL & WIESEL, but one might expect to find hypercomplex units there of perhaps even greater complexity. It is of some interest that HARA (1962) found that cats with the middle suprasylvian gyri removed bilaterally were impaired in discriminating squares from rectangles of different sizes. This is the sort of defect that one might expect in the absence of hypercomplex units.

As units become more complex and deal with larger areas of visual field one would expect to find less topographic localisation. This may be the explanation for its absence in the middle suprasylvian gyrus.

It is not known if perception occurs in a series of stages as the image is projected and progressively analyzed from Visual I to Visual II to Visual III and then perhaps to the suprasylvian gyrus. With this scheme one can imagine the suprasylvian gyrus of the cat handing on an abstract of the visual scene such as "mouse; bearing-latitude x^{o}, longitude y^{o}" for execution by the motor areas of the brain. The alternative is for all the areas to be consulted about matters in which they are specialised. Thus when the cat looks at a square the hypercomplex units at the corners can give most of the information but if it wants to know if the corners are connected, it may have to ask the simple units in Visual I. In the former scheme one would expect severe deficits after removing the middle suprasylvian gyrus. This is not the case (HARA, 1962) although a more severe deficit might have been found if the posterior suprasylvian gyrus had also been removed. A projection from the lateral gyrus to the anterior sigmoid gyrus suggested by the electrophysiological experiments of IMBERT et al. (1966) which has received a little

confirmation in this study (see p.54), is in favour of the second scheme.

A projection to the anterior sigmoid gyrus from the middle suprasylvian gyrus has been described. One might expect this projection to be concerned with visuo-motor co-ordination. However, there are numerous projections to the subcortex (CRAGG, 1965) which might be indirectly concerned.

The projection from the mid-part of the length of the middle suprasylvian gyrus to the cingulate gyrus (CRAGG, 1965) has received some confirmation. As the cingulate gyrus is one of the three regions projecting to the hippocampus, which has been implicated in memory mechanisms, it is conceivable that this is the pathway concerned in the formation of memories, see DRACHMAN & OMMAYA (1964). This receives some support from WARREN, WARREN & AKERT (1961) who found impairment in original learning but not retention of an 'umweg' problem after bilateral removal of the middle suprasylvian gyrus.

Certain similarities are apparent between the cat and monkey visual systems at the cortical level, see Fig.28.

Firstly, although this is speculation, there may be similarities between the striate and adjacent areas. Both animals have an area 17 containing its topographically organised Visual I with the vertical meridian forming most of the perimeter and the area for central vision at the centre of the perimeter. Outside the perimeter is a rim of cortex connected to adjacent parts of area 17 and with callosal connections to the corresponding point in the other hemisphere. This rim broadens at the area for central vision and then some distance from area 17 forms two fingers. In the cat these fingers extend along the length of the lateral sulcus coinciding with the lateral extremes of Visual III, i.e. this is the Visual III vertical meridian with callosal connections. In the monkey MYERS has called

these fingers area 18 proper; they also have callosal connections, and have connection with the rim and therefore presumably with the projection of the vertical meridian.

In the cat there is electrophysiological and anatomical evidence for two representations of the visual field (Visual II and Visual III) between these regions with callosal connections. Visual II and Visual III abut along a line corresponding to the horizontal meridian.

This makes one wonder if the same might not be true of the monkey. There is no electrophysiological evidence for a Visual III but in the squirrel monkey COWEY (1964) has found part of Visual II. Response amplitudes were small and could not be traced further. The anatomical evidence presented by MYERS (1965) is only in abstract. He states "Those neurones located closest to its horizontal meridian project to the furthermost borders of striate receptive area 19" i.e. close to area 18 proper - the finger containing the representation of the vertical meridian. "As the chosen points of area 17 are located farther from the horizontal meridian, the areas of projection onto striate receptive area 19 approach closer to juxtastriate area 18" i.e. the rim around area 17 which is the vertical meridian. Unfortunately this description confuses the issue as what is required is a description of the lesion and its degenerated projection related to both the vertical and to the horizontal meridians. Only in this way are the results meaningful.

In view of this doubt further work is required to establish full details of the monkey projection from area 17, to discover if it is as MYERS (1965) describes, or if there is a Visual II and Visual III as in the cat. In association with this projection system one might expect an increase in the complexity of the receptive fields of cortical units in these areas. It is possible that with increasing complexity precise topography becomes less important so that it

may become impossible to demonstrate highly localised projections to these areas anatomically.

Certain similarities are also apparent between the cat lateral half of the middle suprasylvian gyrus and the monkey infero-temporal region. (See fig 28.)

1. Both receive projections either directly from the striate cortex or the adjacent region. (Monkey - KUYPERS, SZWARCBART, MISHKIN & ROSVOLD, 1965).
2. Both receive projections from the pulvinar (Monkey - CHOW, 1950; SIMPSON, 1952).
3. In the cat there is some evidence (GAROL, 1942) that the anterior commissure interconnects the superior and anterior part of the posterior ectosylvian gyrus and the immediately adjacent part of the middle suprasylvian gyrus. In the monkey the anterior commissure interconnects the middle temporal gyri. (WHITLOCK & NAUTA, 1956).
4. Some visual discrimination deficits have been described after bilateral removal of the middle suprasylvian gyrus of the cat (WARREN & SINHA, 1957; HARA, 1962). Deficits in original learning have been described by WARREN et al. (1961). Other modalities have not been tested.

Recently MISHKIN (1965) has reviewed the effects of lesions in the monkey infero-temporal region. A monkey trained to discriminate between visual patterns will forget the problem and have greater difficulty in re-acquiring them after a bilateral infero-temporal lesion. It is of particular interest that the impairment found by MISHKIN & HALL (1955) in the monkey is in the ability to discriminate between circles of different diameters. This test is very similar to the one used by WARREN & SINHA, 1957 and HARA, 1962, who found deficits in the cat lacking the middle suprasylvian gyrus.

There is no field defect. The deficit is not found in other modalities in the monkey.

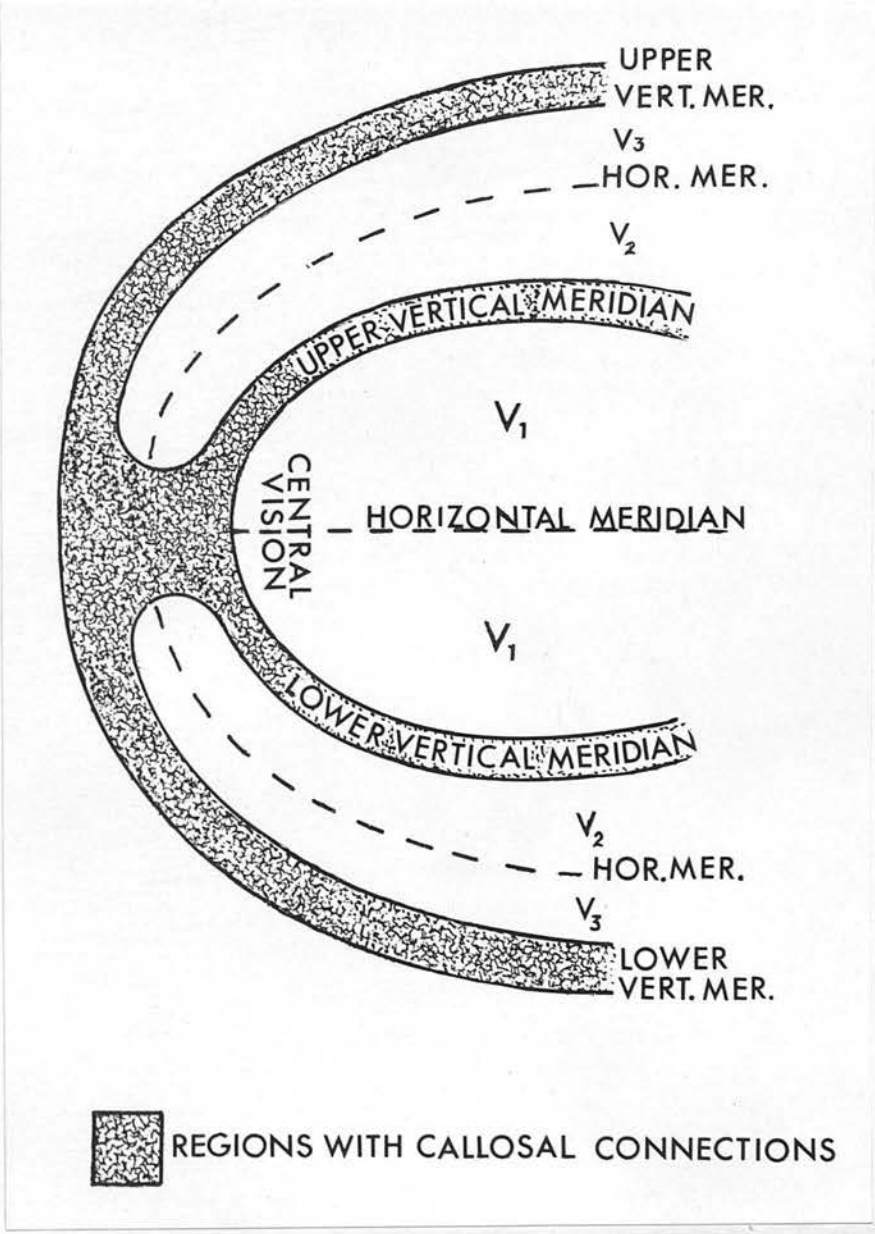

Fig. 28. A schematic diagram to show the possible similarities between the cat and monkey visual systems.
V_1 = Visual area I = Area 17 in cat and monkey.
V_2 & V_3 = Visual areas II and III which have been established in cat but not monkey.
Areas with dense callosal connections have been indicated.

APPENDIX.

The Nauta method adapted for a multi-compartmented holder.

The holder was a sieve made by drilling 22 holes each 7/8" in diameter through a disc of perspex 1" thick and 5½" in diameter. A handle was attached so that the horizontal disc could be moved from one polyethylene dish to another. The lower face of the disc was softened by soaking in acetone, and then pressed flat on a sheet of nylon netting till dry. The perspex that embedded the nylon netting was protected by a coat of Araldite or DePeX (Gurr). Successive sections were placed in the 22 compartments while the sieve was in distilled water, andthis batch of sections was then processed as a unit by moving the sieve from one dish to the next. The dishes contained 100 ml. of the appropriate solutions and were agitated throughout the process by a mechanical rocker.

1. Wash in distilled water - 5 mins.
2. 0.5% Phosphomolybdic acid - 25 mins.
3. Wash - 3 mins. Distilled water.
4. 0.05% potassium permanganate - 12 mins.
5. Distilled water - 3 mins.
6. Decolourizer until colour changes to pale blue.
   ½% hydro-quinone.
   ½% oxalic acid.
7. Three successive washes in distilled water of 4 mins each.
8. Freshly filtered silver nitrate 4.5% - ½ hr.
9. Ammoniacal silver nitrate solution is prepared from :-

Solution A.	Solution B.
1.2 Gm sodium hydroxide.	4.5% silver
50 ml. distil. water.	nitrate.
60 ml. 0.88 ammonia	
330 ml. ethyl alcohol.	

Approximately 40 ml. of solution A are taken to 60 ml. of solution B. One section is removed from the holder and stained in this solution for 2 mins. and then put into reducer. It is examined under the microscope. If it is not satisfactory more of solution A or B is added to produce the desired effect. Once this is correctly adjusted the whole batch of sections in the holder is taken through.

10. Reducer - 2 mins. 0.5 Gm. citric acid.
    5 ml. formalin
    2 l. distilled water.
11. Distilled water 1 min.
12. 1% sodium thiosulphate - 1 min.
13. Three washes of distilled water.
14. Dehydrate through alcohol and mount from beechwood creosote oil. Blot dry and cover with DePeX (Gurr).

Nissl stain.

1. Sections mounted on gelatinised slides and blotted.
2. Formol-alcohol (1:9) - 20 mins.
3. Absolute alcohol.
4. Xylene - 2hrs.
5. Absolute alcohol, down graded alcohols and wash.
6. 0.5% aqueous cresyl violet (pH 3 - 4) - 30 mins.
7. Wash.
8. Differentiate in 70% alcohol until white matter clear.
9. Absolute alcohol, xylene and cover with DePeX.

Weil stain.

Sections mounted as described under Nissl stain, fixed with formol-alcohol.

1. Wash.
2. Leave overnight in 4% iron alum.
3. Wash in distilled water.
4. Stain in freshly prepared stain at 56° C for ½ hr.

   10% haemotoxylin solution in alcohol - 10 ml.
   Saturated aqueous solution lithium carbonate - 2ml.
   Distilled water to 100ml.
5. Wash in running tap water for 10 mins.
6. Differentiate in 4% iron alum.
7. Wash, dehydrate in alcohol, xylene, cover with DePeX.

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